HEALTH RISK ASSESSMENT OF MERCURY CONTAMINATION IN THE VICINITY OF ICI FOREST PRODUCTS CORNWALL, ONTARIO

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HEALTH RISK ASSESSMENT

OF MERCURY CONTAMINATION

IN THE VICINITY OF ICI FOREST PRODUCTS

CORNWALL, ONTARIO

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Report prepared for:

Ontario Ministry of Environment and Energy

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Summary Conclusions

- Exposures to inorganic or total mercury for typical study area residents, integrated through all pathways, are below existing health criteria and are not anticipated to result in adverse health effects.

- Future exposures following closure of the ICI mercury cell room are not predicted to result in adverse effect. Modelled exposures under current and future conditions are not significantly different from those of other persons in Ontario.

- Persons living in the study area will have had mercury exposures somewhat greater than those of the general population, but would have received the majority of this exposure from background diet and not from site-related contamination. Estimated exposure for recreational anglers is greater than any site-related exposures. Adults who engage in recreational angling as well as consume backyard vegetables, will have the highest overall exposures.

- Consumption of contaminated backyard vegetables may potentially lead to notable increases in exposure particularly at the highest measured concentrations, and therefore, this exposure should be avoided for young children. For future consumption, exposure scenarios suggest that both children and adults can safely consume normal amounts of these background vegetables provided that they are thoroughly washed during preparation to remove dirt particles.

Environmental monitoring and the need for risk assessment

Soil and vegetation in the area surrounding the ICI Forest Products Inc. chloralkali plant (formerly CIL) in Cornwall, Ontario has been regularly monitored since 1976, with particular emphasis on the question of mercury contamination resulting from atmospheric emissions from ICI's cell room operations. These monitoring results are summarized in a variety of survey reports and are discussed throughout this report.

In the summer of 1993, residents in the area participated in a survey of

backyard garden vegetables. Mercury levels were measured in various sampled vegetable types. The results indicated higher than normal mercury levels in a number of samples. On the basis of this information, the Ontario Ministry of Environment and Energy (MOEE) and the local Medical Officer of Health cautioned residents to avoid eating homegrown vegetables. Furthermore, surveys of the area conducted in 1987 through 1991 indicated that surface soil and vegetation concentrations for mercury exceeded the Phytotoxicology Upper Limit of Normal (ULN)[1] guidelines and soil decommissioning guidelines for mercury in a number of sampling locations. These guidelines do not represent maximum desirable or allowable levels of contaminants, but rather serve as levels which, if exceeded, prompt further investigation on a case-by-case basis to determine the significance, if any, of the above-normal concentrations. The Standards Development Branch was requested to undertake such an investigation.

This risk assessment's objective is to examine, in some detail, the possible human health implications of exposure to elevated levels of mercury in various media (soil, vegetation, air) in the vicinity of the ICI industrial facility. The most current toxicological information regarding mercury and multi-pathway modelling of human exposure is utilized. The quantitative risk assessment methods described below are used to estimate the potential intake of mercury from soil, backyard vegetables and air and to assess them against other common exposure pathways such as food and drinking water. The implications for human health from exposure to elevated levels of mercury in this area had not previously been fully examined. The results of this risk assessment will contribute to defining the need for future actions, if any.

This report represents an assessment of risk utilizing accepted exposure modelling methods. It is multimedia in nature; that is to say total exposure via a number of possible pathways is examined as well as information regarding typical mercury exposures for persons residing in Ontario. For the purposes of clarity, it is important to distinguish between this type of risk assessment and a community health study. A risk assessment utilizes environmental monitoring data and information about the characteristics of personal exposure to predict the likelihood of an adverse effect occurring.

[1] The Phytotoxicology ULN contaminant guidelines represent the expected maximal concentration in surface soil, foliage (trees and shrubs), grass, moss bags, and snow from areas in Ontario not exposed to the influence of a point source of pollution. The ULN for mercury is 0.5 µg/g (urban soils) and 0.15 µg/g (rural soils). See Appendix A for more information on the ULN.

A community health study would encompass an epidemiological evaluation of the actual exposure and health status of individuals in the area and might include such measures as blood sampling and hair analysis.

Forms of mercury and routes of exposure

Mercury is a metal that occurs naturally in the environment in several forms. In the metallic or elemental form, mercury is a shiny, silver-white, odourless liquid familiar to anyone who has used a mercury thermometer. Some evaporation of metallic mercury occurs at room temperature to form mercury vapour, a colourless, odourless gas. Mercury can also combine with other elements, such as chlorine, sulphur, or oxygen, to form inorganic mercury compounds or "salts". In the following report, both metallic mercury (liquid and vapour) and inorganic mercury compounds are included under the generic term "inorganic mercury". Mercury can also combine with organic compounds to form predominantly methylmercury.

Mercury emissions from the ICI Forest Products, Inc. cell room are in the elemental form as vapour and may also consist of some smaller amount of particulate. However, the form of mercury found in the environment can be changed slowly by micro-organisms and natural processes. Metallic mercury vapour may be changed into other inorganic forms, such as mercuric chloride, and inorganic forms may be changed into organic forms of mercury (and vice versa). No speciation data regarding the various forms of mercury in the soil and vegetables sampled from the study area was available. However, the form of mercury present in soil and vegetation is likely to be principally in the elemental and inorganic forms. Mercury in soil will be present largely as mercuric salts such as mercury sulphite. Therefore, for the purposes of assessing risk, the total mercury measurements in soil, vegetation, drinking water and air samples are treated as inorganic mercury.

Because mercury occurs naturally in the environment, everyone is exposed to very low levels of mercury in air, water and food. However, some people may be exposed to higher levels of mercury. One of the most likely ways that the general population will be exposed to higher levels of mercury is through eating fish or shellfish contaminated with methylmercury. Another most likely form of exposure is though absorbing mercury vapours released from dental fillings. Most silver-coloured dental fillings are about 50% metallic mercury and slowly release small amounts of mercury vapour.

When a substance like mercury is released from an industrial facility such as ICI, it enters the environment. This release does not always result in exposure. In order for exposure to occur, an individual must come into contact with the substance in question. In this situation, exposure to mercury may occur through breathing mercury in air, inadvertently ingesting contaminated soil/dust particles (particularly prevalent in young children), skin contact with soil and consuming backyard vegetables which contain mercury. In addition, exposure may occur through pathways not connected with the contamination in the study area, in particular though the local food basket, through drinking water, and for certain individuals, through consumption of sport fish. It is important to consider these other exposures together with site-specific exposures.

Toxicological effects of mercury

The types of effects which are seen upon exposure to mercury depend upon the form of mercury a person is exposed to and the level of this exposure. Exposure to high enough levels of metallic, inorganic or organic mercury can permanently damage the brain, kidneys and developing fetus. The nervous system is very sensitive to mercury's effects. The changes that mercury causes in the brain are not specific for one type of brain function. Therefore, a variety of effects may occur such as personality changes, tremors, changes in vision or hearing, and difficulties with memory. Not all forms of mercury are equally able to affect the nervous system because of differences in the distribution of different forms in the body. The kidneys are also very sensitive to mercury. All forms of mercury are able to cause kidney damage if large enough amounts enter the body. Recovery from the kidney effects of mercury is likely, however, once the body clears itself of the contamination. Other effects such as immunological, skin disorders and blood pressure have been associated with mercury exposure.

The extent and type of toxic effect is dependent on the form of mercury that people are exposed to. For the purposes of this assessment, which looks at the potential effects of long-term low level exposures to mercury, the critical toxicological endpoints for inorganic mercury are considered to be renal effects, while for methylmercury, neurological effects are considered most significant. The developing fetus and young children are considered to be particularly vulnerable to the effects of mercury and therefore young children are considered as a special exposure group in this risk assessment.

When individuals are exposed to a substance such as mercury, many factors will determine whether harmful health effects will occur and what the type and severity of these health effects will be. These factors include the intake (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking or skin contact), and your individual characteristics such as age, gender, nutritional status, family traits, lifestyle, and state of health.

Technical findings of this risk assessment

Potential mercury exposures in the study area are considered on the basis of currently available monitoring data. Particular higher risk exposure scenarios are also analyzed. These include those residents who may grow and consume their own backyard vegetables, those who may be recreational anglers (i.e. they have a higher level of exposure to methylmercury from consumption of sport fish), and those who may eat both sport fish and garden vegetables. Because of the results of the garden survey which indicated contamination of backyard vegetables in this area, and the existing public health warning against consuming these vegetables, this pathway is modelled in detail, examining both short term (acute) and long term (chronic) consumption of these vegetables. The analysis also attempts to shed light on the change in exposure following the projected closure of the cell room in March 1995 and its significance in terms of potential health risk.

1. Past exposures based on 1990s monitoring studies

(i) Expected exposures for typical Ontario residents

- Exposure to inorganic and methylmercury occurs for all persons in the general population through its presence in very small amounts in air, soil, dust, food and drinking water. Chronic total mercury intakes for the typical Ontario resident (i.e. not living in the study area) are calculated to be 0.27 µg/kg/day[2] for children, with 92% of exposure attributed to inorganic mercury. Average intakes for adults are lower at approximately 0.14 µg/kg/day of which 85% is attributed to inorganic

[2] In this risk assessment, mercury intake is measured in micrograms (µg) per kilogram per day. A microgram is one millionth of a gram.

mercury. By far, the greatest contribution to total mercury exposure is food which accounts for roughly 98% of total exposure in typical Ontario adults and young children.

(ii) Expected exposures for typical residents of the study area

- Estimates of typical exposures for residents in the study area suggest an increased exposure to inorganic mercury relative to the general Ontario population of roughly 1.3 times for the average child, and 1.4 times for the average adult. The chronic mercury intake for the typical community resident is estimated at 0.34 µg/kg/day for children and 0.19 µg/kg/day for adults. This represents an increase of 0.07 µg/kg/day and 0.05 µg/kg/day above typical Ontario resident exposures, for children and adults, respectively.

- By far, the greatest relative contribution to mercury exposure in the study area, as in the general population, is diet. For the typical resident, diet contributes between 70-80% of the total inorganic mercury exposure estimate. Total combined contribution of site-specific media to exposure (ingestion of soil, dermal contact with soil and inhalation of ambient air) is a relatively small fraction of total exposure, accounting for roughly 1/4 of estimated exposure in children and 1/3 of estimated exposure in adults.

- Typical inhalation exposures are estimated at approximately 0.047 µg/kg/day and 0.036 µg/kg/day in adults. Measured ambient air concentrations average 127 ng/m^3 and range from 3.7 to 270 ng/m^3. This exposure is about 10 fold higher than might be seen in a typical Ontario city, and accounts for approximately 15-20% of total of total mercury intake.

- Dermal contact with contaminated soil may contribute slightly to overall total intake of mercury. According to the exposure model, the estimated intake from dermal contact with soil is about 1/2 of the inhalation intake and about 1/10 of the oral intake associated with general diet. It is unlikely that significant amounts of mercury would be absorbed through the skin because of the binding of the mercury to soil particles.

iii) Backyard Vegetable Consumption

- A number of residents in this area grow and consume backyard vegetables. The consumption of backyard vegetables results in a mean increase to the total mercury intake of 0.04 µg/kg/day and 0.01 µg/kg/day in children and adults, respectively, based on the chronic exposure model. This model assumes that consumption of these vegetables in averaged over the entire year.

- In actuality, backyard vegetables are consumed in large amounts on a periodic basis, particularly during the summer and fall season of harvest. Acute periodic intakes in inorganic mercury through backyard vegetable consumption would, on average, add 0.15 µg/kg/day in children, and 0.09 µg/kg/day in adults, and under worst case concentrations, as much as 0.52 µg/kg/day in children and 0.33 µg/kg/day in adults. Under these assumptions, modelling suggests that there is some potential for periodic intakes through eating vegetables that could increase total daily exposures to inorganic mercury by as much as 2 to 3 times compared to those living in the community who do not consume homegrown vegetables. For various reasons, the worst case scenario does not represent the likely exposure for an average child. For the most part, very young children do not consume large amounts of backyard vegetables, and when these vegetables are consumed, it is done periodically.

iv) Ingestion of locally caught fish

- Consumption of angled fish would increase the total mercury intake for the typical adult community resident by 0.18 µg/kg/day, based on calculations using data from the Sport Fish Contaminant Monitoring Program. Consumption of angled fish would increase the total mercury intake for the typical child living in the community by 0.049 µg/kg/day. 80% of this intake is assumed to be in the form of methylmercury and 20% in the form of inorganic mercury.

2. Predicted current and future exposure (post-closure scenario)

- Key matters raised by the closure of the mercury cell-room are the potential for mercury exposure following the closure and the potential health risks, if any associated with these exposures. For this reason, a plausible scenario of multimedia exposure was modelled which included off-gassing of mercury form soil to air and uptake of mercury form soil into vegetables. Estimated total mercury intakes for residents under this scenario are not significantly different from those of the general Ontario population at 0.30 µg/kg/day for children and 0.16 µg/kg/day for adults.

- A significant decrease in mercury concentrations in ambient air is predicted due to closure of the mercury source. However, some off-gassing of mercury vapour from soil to air is still likely to occur. Ambient air levels following closure of the cell room facility are estimated to be 2-9 ng/m^3 on the basis of soil-to-air emission modelling. These levels are well within the typical range of urban air quality for mercury.

- There is also no significant exposure associated with backyard vegetable consumption as calculated, based on limited uptake of mercury from soil into plants and a significant reduction of aerial mercury deposition.

- Following closure of the cell room facility, it is estimated that mercury in soil will decrease over time and be modelled to background concentrations through natural degradation processes within 12-20 years. This estimate is based on the modelled source emission rates from soil together with assumptions regarding the depth of contamination. Correspondingly, exposures should generally decrease over this period. Field studies of soil depth profiles would be required to verify these assumptions.

3. Health Implications

In carrying out this assessment, current numerical toxicological criteria were gathered from a number of leading agencies including the U.S. Environmental Protection Agency (EPA), the World Health Organization (WHO), the Agency for Toxic Substances and Disease Registry (ATSDR)

and Health Canada. Such values exist for inorganic mercury, methylmercury and total mercury and are usually specific for inhalation or oral routes of exposure. As such, predicted exposures via individual routes are examined along with total exposure from all routes.

(i) Total Exposures in Study Area

- The estimated mercury exposures associated with levels of mercury in soil and air, as measured in recent surveys, do not suggest a potential for undue exposure or adverse health effect in this community related to mercury. Previous backyard vegetable contamination may have resulted in short-term increased exposures in some individuals, notably young children. These exposures would not appear to have presented any imminent health hazard. However, it cannot be categorically determined that increased short term exposures due to consumption of backyard vegetables would not be without some small degree of additional risk (see discussion below).

- Examining intake of total mercury, typical exposures for children and adults within this community are well below the permissible tolerable weekly intake set by the WHO.

- All measured and modelled soil concentrations of total mercury fall well below the new MOEE decommissioning guideline for mercury in residential soil (10 ppm). As this is a health based guideline, this observation underlines the above conclusion.

- A thorough review of the scientific literature revealed no reported community health problems associated with airborne mercury emissions from this type of industrial facility.

(ii) Exposure through ingestion

- Total exposure via ingestion for a typical child is close to, but does not exceed, the U.S. EPA oral reference dose for inorganic mercury. Additional exposures above the typical exposures therefore must be closely examined.

- The total child and adult exposures via ingestion including background dietary exposure are well below (less than 10%) of the ATSDR

minimum risk level of 2 µg/kg/day, as well as below the U.S. EPA oral reference dose of 0.3 µg/kg/day. Under a worst case assumption of maximum measured soil concentration, ingestion exposure is only marginally increased and is still below the most conservative health criteria for oral exposure.

(iii) Skin contact

- Estimated dermal exposures are small and are not anticipated to result in dermatological effects which are generally associated with much higher exposures such as would be encountered in an occupational setting. Thus, exposures associated with soil in this area do not constitute an undue health risk.

(iv) Exposure through inhalation of air

- The average ambient air concentration and maximum concentrations are below the inhalation chronic acceptable exposure level set by the California Air Toxics Hot Spots Program. All values are also significantly below the reference concentrations suggested in the U.S. EPA HEAST Guidelines, utilized recently in the Ministry's assessment of the air quality in Windsor. The calculated intakes are several hundred times lower than the WHO human inhalation exposure equivalent of a lowest observed adverse effect level for renal effects. In summary, adverse health impacts are not anticipated at these air levels for the following reasons: *1.)* comparison to the 1991 WHO human effects equivalent for renal effects; *2.)* the large numerical adjustments (safety factors) in derivation of the MRL values (at least 100 fold); *3.)* air levels are within the 1993 California air guidelines; *4.)* air levels are within the existing U.S. EPA inhalation reference dose (although this is currently under review); *5.)* total exposures are within acceptable criteria for total exposure by all routes; and *6.)* inhalation exposure is less than 1/5 of total exposure and significantly less than normal dietary exposure. The interpretation of these findings is somewhat complicated by the current changes in air related health criteria (agencies develop health criteria based on different assumptions and slightly different data sets).

(v) Backyard Vegetable Consumption

- On average, the chronic intake of mercury through consumption of vegetables would increase the chronic daily intake from all dietary sources in children to marginally below the U.S. EPA oral reference dose for ingested inorganic mercury. Adult exposures would also remain below this health criteria. A maximally exposed child, assuming the very highest measured mercury concentration in vegetables, may have some potential for exposure slightly greater than the chronic oral ingestion reference dose, although this amount of exposure would not generally be expected to be the case. It should be noted that not all of the vegetables for which data were available (beet top, beet root, tomato and lettuce) would be the types of vegetables most commonly consumed by very young children and would unlikely be consumed in large amounts. The highest mercury concentrations by far were seen in beet tops, followed by lettuce. This is indicative of the potential for leafy vegetables to accumulate aerially deposited mercury emissions. Root vegetables had much lower mercury levels and therefore mercury exposure resulting from their consumption is likely to be much lower. Therefore, the estimated highest exposures, either chronically or shorter-term are atypical and would not necessarily apply to any children unless the exposure assumptions are met for these worst cases.

- Modelled periodic shorter term exposures are well below the ATSDR minimal risk level for acute oral exposure and intermediate exposure. Therefore, it is unlikely that these exposures constitute an imminent health hazard. On the other hand, the additional intake provided by backyard vegetable consumption for those consuming vegetables at the maximum reported concentration could result in exposures which exceed the WHO permissible weekly intake for total mercury. Again, these would be atypical exposures, assuming that, in pre-school children, 98 g of these vegetables were eaten per day each day over a one week period. Also, the WHO guideline is based primarily on restricting methylmercury contact and does not apply when additional mercury intake is the inorganic from. Overall, these findings suggest that backyard vegetable consumption during the years of cell room emissions may have resulted in some increase in mercury intake for some individuals but that these exposures would not appear to have presented an imminent health hazard, unless consumed at much higher levels than assumed. It cannot be categorically determined that

these short term exposures would not be without some additional risk. However, these are unlikely to be large, given that on a chronic basis backyard vegetable consumption accounts for only 11% total exposure in children and are approximately 1/6 of the intake compared to background diet intake. Also, there is a very large margin of safety (uncertainty factor of 1,000) contained within the U.S. EPA reference dose derivation. On balance, it would appear that although the additional exposure associated with eating vegetables is unlikely to be associated with adverse effects in children, prudence would suggest that this exposure be avoided under circumstances of continuing emissions from the ICI facility.

(vi) Current and future exposures

- No adverse health implications are anticipated with projected exposures following cell room closure in this community. Exposures essentially will not differ from those of other Ontario residents. There is also no significant exposure associated with backyard vegetable consumption as calculated based on the uptake of mercury from soil into plants; adults and children should be able to safely consume vegetables in a normal fashion. As is always the case, thorough washing of vegetables will ensure reduction of residual mercury concentrations in those foods, and this will decrease intake further. Projected ambient air concentrations are well below even the most stringent inhalation reference concentrations for health criteria. The assumptions regarding mercury concentrations in the area following closure of the cell room facility, however, can only be verified through additional environmental monitoring.

- The exposure scenario associated with highest exposure (largely methylmercury) is the consumption of locally caught sport fish. Recent studies of mercury levels in hair and blood in Cornwall-area fish eaters did not reveal any individuals with mercury levels exceeding Health Canada guidelines. Inorganic mercury was undetectable in most samples. Although it was not known at the time of this report whether any residents of the study area were participants in this study, this is possibly corollary evidence of no significant exposures in the study area.

Recommendations

1. MOEE should undertake limited monitoring of mercury levels in soil and backyard vegetables in this area in order to allow verification of assumptions regarding current and future mercury exposures. Further, at-depth soil profiles should be determined in the most contaminated zones to verify assumptions in the calculation of soil-to-air emissions and the projected reduction in mercury levels in soil over time.

2. MOEE should undertake a limited mobile TAGA survey to confirm post-closure reductions in ambient air levels of mercury within the study area itself.

3. Young children should not consume any backyard vegetables grown prior to 1995 which may be stored (e.g. canned, pickled, frozen), including any prepared foods containing these vegetables (e.g. frozen soups). However, for the current growing season and in the future, no significant exposure to mercury is projected due to closure of the cell room. Therefore, residents may choose to consume backyard vegetables freely. However, thorough washing (and peeling, where appropriate) of homegrown produce is emphasized in order to remove any soil particles and thereby minimize the possibility of exposure.

4. This Ministry, together with local public health officials, should endeavour to annually advise residents in the community of means to reduce mercury exposure.

PREFACE

It is important to understand that there are a number of uncertainties associated with the process of risk assessment arising from the lack of empirical scientific data in numerous areas. The risk assessment approach presented utilizes assumptions which are generally considered conservative predictive methods. Actual risks may in fact be much lower than those described, although this cannot be precisely determined. For these reasons, risk estimates should be considered not as true or actual risks but as estimates to be utilized as a crude measure of potential impact. The risk assessment, herein, is specific to the site in question and should not necessarily be directly applied to interpretation of other situations or scenarios.

In preparation of this report, every attempt was made to obtain and utilize the most current toxicity information available. To this end, staff of the Standards Development Branch attended the International Neurotoxicity Conference which dealt specifically with mercury neurotoxicity and effects of low-level exposure. At this conference, staff acquired valuable new information which has bearing on this assessment.

1.0 INTRODUCTION

1.1 The health risk assessment process

In assessing environmental health risks, whether in a site-specific or general population context, it is increasingly recognized that an integrated assessment of exposure through multiple pathways is necessary to understand the total risk to populations as well as the contributing risk posed by each media under examination. The general methodology used is based on conventional models of risk assessment (see Figure 1.1). The risk assessment consists of four principle components. These are: *1.)* hazard identification (toxicological profile); *2.)* dose-response assessment; *3.)* human exposure assessment; and *4.)* risk characterization. This risk assessment framework is aimed at determining what the magnitude of exposures may be through various pathways for different subgroups and whether or not adverse or undesirable effects from such chemicals would be expected from such exposures.

1.1.1 Hazard identification

Hazard identification is the stage of risk assessment in which preliminary identification of the potential adverse health effects from a chemical occurs in order to allow preliminary judgement regarding the level of concern posed by the chemical in question.

The hazard identification process identifies qualitatively the type of adverse health effect (e.g. cancer, neurotoxicity) associated with the chemical in the scientific literature. This can include animal toxicology studies, epidemiological studies, and where data are lacking, a comparison of the chemical's structure/activity to that of substances already known. The toxicological information provided in this risk assessment is not meant to represent a comprehensive review of all the available information, but rather a synopsis of the more current and relevant summary information. Information sources from which toxicity data were obtained include computerized databases such as the Registry of Toxic Effects of Chemical Substances (RTECS, 1987), TOXLINE, Hazardous Substances Data Bank (HSDB), United States Environmental Protection Agency (U.S. EPA) Integrated Risk Information System (IRIS, 1990), Chemical Evaluation Search and Retrieval System (CESARS) and the Chemical Abstract Service (CAS). Other information sources included review and regulatory documents

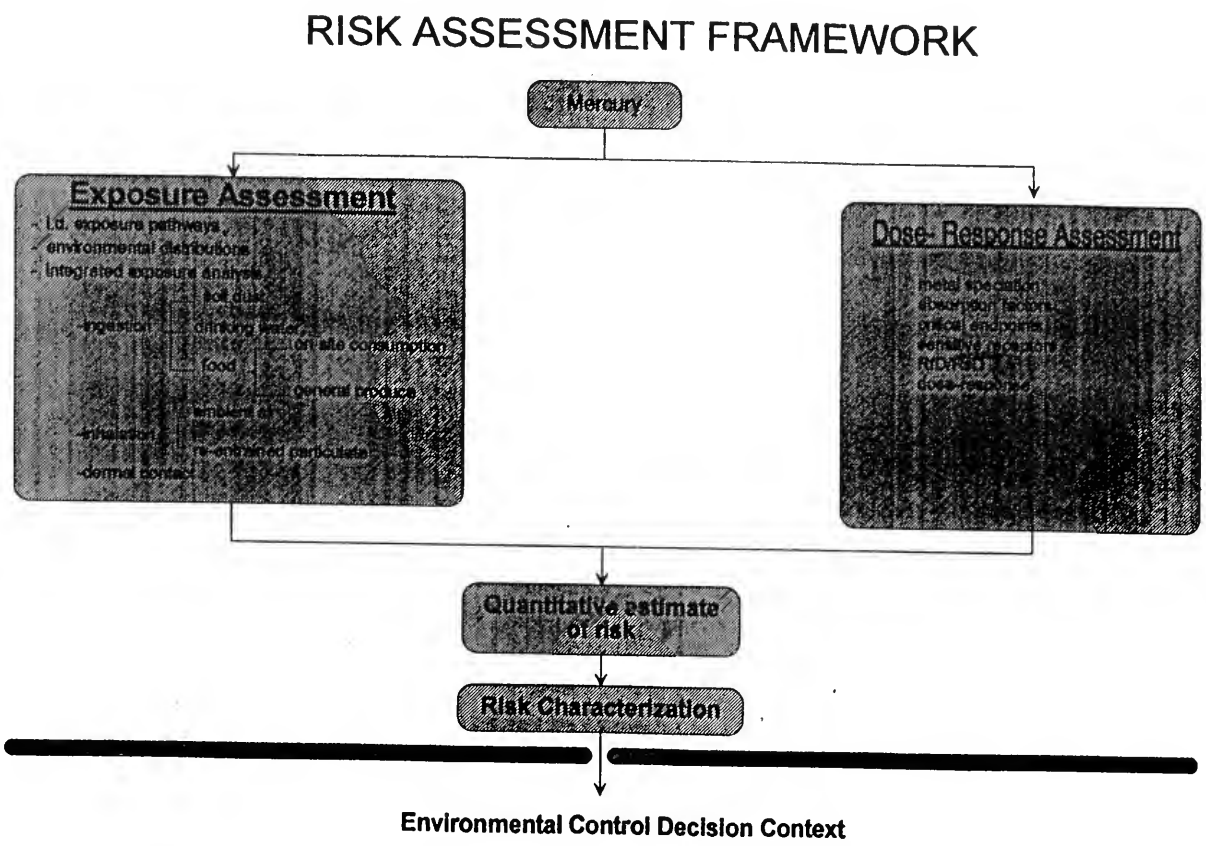

Figure 1.1: Risk Assessment Framework

from this ministry and other agencies and where indicated, the original scientific literature.

1.1.2 Dose-response assessment

Dose-response assessment is the determination of the relationship between the magnitude of exposure routes and the probability of occurrence of toxic effects on human and non-human biota. For non-carcinogenic effects, dose-response information is generally utilized by regulatory agencies to develop acceptable exposure levels (e.g. acceptable daily intakes (ADI) and reference doses (RfD)) below which adverse effects are not expected to occur.

In quantitative risk assessments for cancer, carcinogenicity is often expressed in terms of a potency slope, which is then used to calculate the probability (risk) of cancer associated with a given exposure level.

In this assessment, there is no attempt to develop dose-response relationships from reported studies; rather this information is adopted from credible regulatory bodies. For both carcinogenic and non-carcinogenic endpoints, potency factors and reference dose values are gathered from various agencies, and a brief description of their basis, where available, is provided. These values have, in general, been subjected to rigorous peer review within the respective agencies.

1.1.3 Human exposure assessment

Exposure assessment is the qualitative and quantitative determination or estimation of the magnitude, frequency, duration and route of exposure to a particular physical, chemical or biological disturbance in the environment. It delineates the major pathways of exposure (e.g, air, water, food), the levels of exposure from each pathway, and the total exposure from all pathways that contribute to the health risk of concern. Data for exposure assessment may be obtained from monitoring studies of the contaminant and from dynamic modelling of its environmental fate.

Exposure assessment extends to an evaluation of the uncertainties associated with the determination or estimation. Individual exposures to a single environmental media should be evaluated in the context of an

integrated multimedia assessment of total exposure.

In this assessment, the following routes of exposure were examined individually:

1.) Ingestion of:
   - *i)* soil
   - *ii)* backyard garden vegetables
   - *iii)* food (background)
   - *iv)* locally caught fish
   - *v)* drinking water
2.) Dermal exposure resulting from contact with contaminated soil
3.) Inhalation

With regards to estimating total exposures (multimedia analysis) five exposure scenarios for both children and adults were evaluated using existing monitoring data:

1.) typical community resident
2.) typical community resident consuming backyard garden vegetables
3.) typical community resident consuming angled fish
4.) typical community resident consuming both backyard garden vegetables and angled fish
5.) typical community resident consuming backyard garden vegetables - post cell-room closure (modelled using monitoring data as its basis)

Furthermore, these exposure scenarios were compared with mercury exposures calculated for typical Ontario residents.

1.1.4. Risk Characterization

In this step, estimates of exposures from single or combined pathways are compared with the information on dose-response and current exposure limits defined from toxicological information. This step includes a determination of the major routes of exposure and of the specific population or part of the environment at risk. The magnitude and type of risk from each route of exposure is assessed, and an evaluation is made of the contribution of the particular route to the overall risk.

Outcomes can include the probability that a particular adverse effect will

occur in a given population, the relative contributions of various pathways to outcomes and qualitative judgements on the potential for health effects to occur.

The output of risk characterization is usually given as a single point estimate of risk. However, it must be realized that some degree of uncertainty is contained in these apparently precise predictions of risk.

The question of uncertainty in risk assessment has been reviewed (Finkel, 1990). The sources of uncertainty include:

- lack of precision of scientific measurements;
- incomplete knowledge of underlying biological and environmental processes;
- variability in human and animal populations;
- assumptions and limitations inherent in predictive models; and
- random workings of chance.

Time limitations in the preparation of this document precluded systematic quantification of the attendant uncertainties of each stage of the risk assessment process. However, at each decision point, assumptions were conservative. This does not mean, however, that worst-case assumptions were always accepted.

2.0 HAZARD IDENTIFICATION

An extensive review of the toxicology, human epidemiology, environmental fate, and properties of mercury was recently published by the Agency for Toxic Substances and Disease Registry (ATSDR, 1994) of the Public Health Service, U.S. Department of Health and Human Services. This review encompasses past and recent findings obtained from a detailed literature search. It provides an excellent integrative and interpretative evaluation of the mercury issue as related to its potential health effects on humans following exposure through various environmental media. Excellent re-evaluations of the quantitative aspects of mercury toxicity have also been developed by the World Health Organization (WHO, 1990; 1991). These documents should be referred to for further detail.

2.1 Absorption and metabolism

Exposure to mercury can occur through a number of sources. Mercury is present in small amounts in all environmental media (soil, air, and water) and is also present in trace amounts in food. As such, exposure to mercury occurs through a number of different pathways: inhalation, ingestion and dermal contact. Inorganic mercury is the predominant form arising from industrial discharges. Consequently, for the purposes of this current health risk assessment, information pertaining to inorganic mercury compounds is most relevant.

In conjunction with the multimedia/multipathway nature of mercury, the various physical and chemical forms of this element may also have different toxicological properties as a result of pharmacokinetic and metabolic differences. In fact, although the toxicity of the various forms of mercury seems to be related to cationic mercury (Hg^{+2}) *per se*, it is the solubility, biotransformation, and tissue distribution of the different species of Hg that, ultimately, will determine the target organs and, possibly, the mechanisms of action. These parameters are influenced by the valence state of mercury and the associated anionic components, when present (Goyer, 1991). Consequently, the assessment of potential health effects induced by environmental mercury must integrate knowledge about the various exposure pathways and the associated chemical and physical forms of the element in these media (speciation).

The toxicity of mercury is dependent on its bioavailability which in turn, is

determined by the chemical form or species of mercury. Elemental mercury, mercury salts and phenyl mercury compounds, although toxic, are excreted efficiently and therefore, trace amounts of such compounds are not usually recognized as an environmental or human health concern. In contrast, alkyl mercury is excreted very slowly in humans and has a half-life of 70 days (Atlas and Bartha, 1981).

Elemental mercury (Hg°) is the predominant form of mercury present in the atmosphere and, consequently, is considered the most relevant for pulmonary exposure. Mercuric (Hg^{+2}) ions and, to a lesser extent, mercurous (Hg^{+}) ions are mainly present in the aqueous phase and may contribute to oral intake of the element. On the other hand, organic mercury, such as the mono- and dimethylmercury forms which can be produced by certain microorganisms, are considered to be among the predominant chemical species of mercury in some foods (*e.g.* fish and fish products) (WHO, 1990).

2.1.1 Absorption by ingestion

The gastrointestinal tract provides a good barrier to elemental and inorganic mercury; consequently, most ingested elemental or inorganic mercury will not be absorbed through the digestive system (U.S. EPA, 1984). Limited data also suggest that oral absorption of inorganic mercury is poor and is estimated at approximately 0.1% for metallic mercury (Hg°), and 7% for an ingested dose of divalent mercury (Hg^{+2}). In contrast to these values, it has been reported that up to 95% of a tracer dose of aqueous methylmercury nitrate was absorbed in humans (ATSDR, 1994; Clarkson, 1992), thus indicating the high lipophilic nature of organomercurial compounds.

There is some controversy regarding the absorption of metallic mercury. Some researchers believe that it is readily absorbed through the gastrointestinal tract based on the observation of elevated blood mercury levels following ingestion of metallic mercury (Merck and Co. Inc., 1983; Suzuki and Tonaka, 1971). However, other observations indicate that occasional accidental consumption of metallic mercury has caused no ill effects (U.S. EPA, 1984). Furthermore, on the basis on animal studies, others believe that it is poorly absorbed by the gastrointestinal tract (Friberg and Nordberg 1973; U.S. EPA, 1984). Absorption of inorganic mercury compounds (HgO and $HgCl_2$) in the gastrointestinal tract of rats was studied by Endo *et al.* (1991) who estimated that 5 to 10% of the administered

mercury dose was absorbed. The U.S. EPA (1984) examined the abstract pertaining to a study by Rahola *et al.* (1973) which used orally administered mercuric acetate bound to calf liver to investigate the amount of mercury absorbed through the gastrointestinal tract in 8 volunteers. The U.S. EPA (1984) reported that 15% of the inorganic mercury was absorbed by the gut and that this value was similar to values reported in experimental animals (Clarkson, 1971). However, upon examining the actual Rahola *et al.* (1973) abstract, this value could not be confirmed. Therefore, the next highest absorption value available in the literature, 10% (Endo *et al.*, 1991) was used for the current exposure assessment.

2.1.2 Absorption by inhalation

There is limited quantitative data on the absorption of metallic mercury vapors (Hg°) by humans after inhalation exposure, although it is the most common route of inorganic mercury uptake (Goyer, 1991). Because of its high lipophilic properties, metallic mercury (Hg°) is readily absorbed (100% absorption of the inhaled vapor) across the alveolar membranes of the lungs and diffuses into blood (ATSDR, 1994). However, no studies were located regarding the pulmonary bioavailability of organic mercury in both humans and animals, although indirect evidence suggest that uptake may be quantitatively important.

WHO (1976) estimated that 80% of mercury that enters the lungs is retained. Neither the basis of this value nor the form of the mercury is specified in this document. Other studies (Teisinger and Fiserova-Bergerova, 1965; Nielsen-Kudsk, 1965; Hurch *et al.*, 1976) have also determined that this value is the amount of inhaled metallic mercury vapour absorbed from the respiratory system by humans. Details of these studies were not described.

2.1.3 Dermal absorption

Little data is available on the dermal absorption of the various forms of mercury although ACGIH (1991) and ATSDR (1992) indicate that it is considered relatively insignificant for ionic mercury, and moderate for elemental mercury. Mercury may be absorbed slowly by skin. Inorganic and metallic mercury can penetrate healthy skin and cause irritation. The absorption of inorganic mercury may also contribute to the systemic effects of mercury absorption via inhalation and ingestion (NIOSH, 1973). Most

cases of dermatitis from mercury exposure have been associated with exposure to organic mercury compounds (NIOSH, 1973; WHO, 1976).

A review of the literature reveals that there is no substantive dermal absorption data on mercury (see discussion of dermal modelling in section 4.2.3). The fraction of mercury absorbed from dermal contact with soil particles is complex to determine. For the purposes of this exposure assessment, an absorption value reflective of some recent dermal absorption data, as well as the binding of mercury to soil (which will hinder absorption) was assumed.

2.1.4 Distribution and elimination

Following its absorption in the systemic circulation, mercury is rapidly translocated to various organs and tissues. As previously mentioned, distribution profiles are influenced by the chemical form of mercury. Ionic mercury has a higher binding capacity to plasma proteins and organomercurial compounds tend to concentrate in red blood cells as a result of their weak binding to hemoglobin. Generally, higher amounts of elemental (Hg°) and organic mercury are transferred through the placenta and blood-brain barriers, while protein-bound inorganic mercury is more likely to be filtered through kidney glomerules and retained in tubular cells (Goyer, 1991). Retention in the brain and the fetus is favored by the oxidation of organic mercury to divalent mercury (Hg^{+2}), which subsequently binds to sulfhydryl and/or thiol groups. Blood-brain ratios in the range of 5 to 1 have been reported in primates, and a comparable range has been suggested for humans (Clarkson, 1992). However, several other oxidation-reduction mechanisms have been shown to occur in tissues, and the influence of enterohepatic cycling and secondary redistribution of water-soluble organic forms of mercury must be accounted for when assessing the pharmacokinetics of this chemical.

The elimination of mercury proceeds *via* three main routes, depending on its chemical form. Inorganic mercury is excreted in the urine and feces, while unmetabolised organic mercury is excreted predominantly *via* the feces, in humans. Small amounts of elemental mercury (Hg°) are also eliminated *via* expired air (ATSDR, 1992; Goyer, 1991). Based on observations of six subjects that have ingested a single low dose of mercury, and on Iraqi mothers exposed for many months, the biological half-life of mercury in blood has been estimated at approximately 50 days (40-105 days) (Clarkson, 1992; WHO, 1990).

2.2 Toxicological effects

Definitive data on the mechanisms of action of mercury and mercury compounds is limited. It is believed that the high affinity and binding of the mercuric cation (Hg^{+2}) to protein-containing sulfhydryl and/or thiol groups could be the main etiologic factor. As previously noted, the adverse health effects induced by mercury compounds are somewhat influenced by the route of exposure.

Inhalation of metallic mercury vapors (Hg°) has been associated with systemic toxicity in both animals and humans. Under acute, high exposure levels (1-100 mg/m^3), respiratory, cardiovascular, neurological, hepatic, renal, and gastrointestinal effects have been demonstrated in animals (ATSDR, 1994). In situations of chronic, low concentration exposure scenarios, however, the major target organs of Hg°-induced toxicity are the kidney and the central nervous system. Low Observed Adverse Effect Levels (LOAELs) for neurological disturbances have been reported to be in the range of 50-100 $\mu g\ Hg/m^3$ in humans. According to the recent ATSDR (1994) summary document, no studies were located on effect levels concerning inhalation exposure to other inorganic mercury compounds (mercuric or mercurous salts, oxides, etc.), and limited information is available for organic compounds.

A substantial amount of information is available on the effects of ingested mercury in humans and experimental animals. As with inhalation exposure to metallic mercury vapor, major target organs of toxicity following oral exposure to inorganic and organic mercury are the kidney (nephrotic syndrome, glomerular and tubular pathologies) and the central nervous system (functional neurotoxicity and neuropathological changes). Oral exposure to mercury, especially to organic mercury forms, has been observed to result in adverse developmental effects in humans and experimental animals.

In this regard, the outbreaks of severe poisoning that occurred in Japan (Minamata Bay) in the 1950's and Iraq in the early 1970's, revealed important characteristics of the toxic action of methylmercury in human adults. Clarkson (1992) summarized these effects as follows: *1.)* overt signs and symptoms usually take weeks or months to manifest; *2.)* all the signs and symptoms are due to selective damage to the nervous system; and *3.)* the brain is the primary target organ as manifested by the loss of neuronal cells in the visual cortex and the granule layer of the cerebellum. The

reasons for these specific effects (latent period and focal damage), however, are not known.

The amount of mercury in blood (from recent and past exposures) may be readily estimated from the concentration of mercury in hair. Further, since pharmacokinetic modeling allows relationships between blood mercury and exposures to be established, a direct quantitative cause-effect relationship for various neurological effects and exposure to mercury is possible. Of major importance for the assessment of neurological effects induced by mercury in humans has been the establishment of dose-response relationships for various end-points. Hence, a dose-related increase between the severity of the effect and the concentration of mercury in hair is available for pathologies progressing from paresthesia to ataxia, dysarthria, deafness, and finally death.

The Iraqi and Japanese outbreaks have also revealed the capacity of mercury to induce prenatal toxicity in humans, an observation that was later confirmed by experiments in laboratory animals. Clinical findings have included reports of infants suffering from severe brain damage where mothers were exposed to methylmercury during pregnancy. Several cases of mental and psychomotor retardation were also noted. Generally, these effects were seen predominantly in male infants, a consequence which most probably seems to be associated with the higher sensitivity of males to mercury-induced mitotic arrest of dividing neuronal cells. Finally, in his recent discussion, Clarkson (1992) indicated that, based on dose-response data, the fetus may be 5-10 times more sensitive than the adult to brain damage from methylmercury.

Finally, some evidence obtained from experimental studies in laboratory animals has also suggested that both organic and inorganic mercury could affect the cardiovascular system (increase in systolic blood pressure and cardiac contractility, decreased baroreflex sensitivity), gastrointestinal system (increased incidence of forestomach hyperplasia in male rats chronically exposed), adrenocortical function (increase in adrenal and plasma corticosterone levels), immunological system (lymphoproliferative response suppression, decrease in antibody response, etc.), and reproductive function (increased incidence of resorption, spontaneous abortions, decreased litter size, etc.).

Of importance for public health is the genotoxic status of mercury. In this regard, no information is reported by the U.S. EPA in their Integrated Risk

Information System (IRIS) database (IRIS, 1994). ATSDR (1994) reports that there is inconclusive evidence that occupational exposure to metallic mercury and to organic and inorganic mercury compounds, primarily through inhalation, causes structural (clastogenicity) and numerical (aneuploidy) chromosome damage in human lymphocytes as observed in occupationally exposed workers. These same conclusions apply to the case of subjects orally exposed to mercury. Therefore, the evidence of genotoxic potential of mercury in humans is inconclusive. Experimental *in vitro* and *in vivo* studies, on the other hand, provide some evidence of the capacity of inorganic and organic mercury to interact with DNA in mammalian and bacterial cells. Recently, WHO's International Programme on Chemical Safety (IPCS), after reviewing the mutagenicity of methylmercury, concluded on the positive genotoxicity of this compound in standardized *in vitro* tests (WHO, 1990).

The U.S. EPA reports that data pertaining to the carcinogenicity of inorganic mercury or its salts in humans and animals is not available in the literature (U.S. EPA, 1984). A two-year bioassay is being conducted by the National Toxicology Program (NTP) on the effects of mercuric chloride on mice and rats is currently undergoing pathological review (NTP, 1993). Data from this study is as of yet unavailable.

There is no evidence from epidemiological studies that indicates inhalation and oral exposure to inorganic and organic mercury produces cancer in humans. However, recent studies conducted by the NTP indicate that lifetime gavage exposure to mercuric chloride ($HgCl_2$) results in an increased incidence of forestomach squamous cell papillomas in low-dosed males, and a marginal increase in the incidence of thyroid follicular cell carcinomas in high-dosed male rats. Furthermore, renal tubule tumors were evident in 3 of the 49 high-dosed male mice. The implications of these findings for risk assessment are not yet clear in view of a lack of dose-response relationship for these effects. Mercury (inorganic) is currently classified in Group D (not classifiable as to human carcinogenicity) by the U.S. EPA (IRIS, 1994) on the basis of unavailable human data, and inadequate animal and supporting data. No information on the carcinogenic potential of mercury has been disseminated by the International Agency of Research on Cancer (IARC) and the NTP.

3.0 DOSE-RESPONSE ASSESSMENT CRITERIA

3.1 Inorganic and elemental mercury

3.1.1 Ingestion

The establishment of acceptable daily intakes (ADI) or reference doses (RfD) for inorganic mercury has been the subject of recent major reviews by WHO (1990; 1991), the U.S. EPA (1984) and ATSDR (1994). The establishment of such values has been hindered by a lack of adequate human data dealing with the relationship between dose and effect. For the most part, these values have been derived from animal studies in which various salts of inorganic mercury have been administered to rodents for various periods of time. It is well recognized that more human research is needed in order to arrive at more exacting determinations for human health risk evaluation of inorganic mercuric mercury compounds at low levels of exposures.

The above agencies have all concluded that the most sensitive adverse effect for mercury risk assessment is the formation of mercuric-mercury-induced auto-immune glomerulonephritis (a kidney effect). The production and deposition of IgG antibodies in the glomerular basement membrane is generally considered the first step in the formation of this form of glomerulonephritis. A particular problem associated with effects such as this one which are allergic and immunotoxic in nature, is that they are partially regulated genetically. They are there for maybe a small fraction of the population that is particularly sensitive to these types of effects. This individual sensitivity has been observed in animal studies. The WHO (1991) has concluded that:

> "A consequence of an immunological etiology is that it is not scientifically possible to set a level for mercury (i.e. in blood or urine) below which mercury-related symptoms will not occur in individual cases, since dose-response studies for groups of immunologically sensitive individuals are not yet available. Species selection or extrapolation from animal data to human effects points to the Brown Norway Rat as the preferential test species for the study of autoimmune effects. Although these effects have also been observed in rabbits, the Brown Norway rat is considered to be the best animal model for the study of mercury induced kidney damage at present."

In calculating a human oral exposure equivalent for a lowest observed adverse affect level (LOAEL), WHO (1991) concluded that a no-observed adverse affect level (NOAEL) could not be determined from animal studies. The LOAEL was determined from a subcutaneous exposure study by Druet *et al.* (1978). Using this animal LOAEL of 0.05 mg/kg, a human oral LOAEL value was calculated as follows:

From the Druet *et al.* (1978) study:

$$\textit{average subcutaneous dose in rats} = \frac{0.05\,mg/kg \times 3\ days \times 0.739}{7\ days}$$
$$= 0.0158\ mg/kg/day$$

where,

0.05 mg/kg= dose of $HgC1_2$ injected subcutaneously into rats (LOAEL)

3 days = number of days per week doses were administered

7 days = number of days per week

0.739 = fraction of $HgC1_2$ that is Hg^{2+} ion

Therefore,

$$\textit{human oral exposure equivalent} = \frac{0.0158\ mg/kg/day \times 70\ kg \times 100\%}{7\ \%}$$
$$= 15.8\ mg/day$$
$$= 0.225\ mg/kg/day$$

where,

7 % is the absorption efficiency via the oral route

The U.S. EPA has previously developed an oral RfD for mercuric mercury compounds of 3 x 10-4 mg/kg/day or 0.3 µg/kg/day, also based on studies using Brown Norway rats, as well as the entirety of the mercuric mercury database. An RfD is a daily intake, within one order of magnitude, below which an adverse effect is not anticipated over a lifetime. As of December

1994, this oral reference dose was pending review by a U.S. EPA working group. The details of the derivation of this RfD are provided in Appendix B. It is based on back calculations from a Drinking Water Equivalent Level (DWEL) recommended to and subsequently adopted by the U.S. EPA Office of Research and Development in October 1987 of 0.01 mg/L, equivalent to 0.003 mg/kg body weight per day (0.3 µg/kg/day). This DWEL is based on the results of three studies - namely, Druet *et al.* (1978), Bernaudin *et al.* (1981), and Andres (1984). The LOAEL exposure levels in these studies varied between 0.226 mg/kg/day to 0.633 mg/kg/day; an uncertainty factor of 1000 was applied. A safety factor of 1000 accounts for: *1.)* lack of a NOAEL in the animal studies; *2.)* possibly sensitive individuals; *3.)* extrapolation from subchronic to chronic data, resulting in an oral RfD of 3 X 10^{-4} mg/kg/day. It is important to recognize the large margin of safety implicit in this derivation.

The confidence in this particular oral reference dose value is considered high by the U.S. EPA based upon their expert review. Two key recommendations stemming from the expert review of this RfD conducted by U.S. EPA (1987) are that:

1. The Brown Norway rat is a good surrogate for the study of mercury induced kidney damage in sensitive humans. Therefore, the uncertainty factor used to calculate criteria in health advisories should be reduced by 10 fold.

2. Divalent mercury absorption values of 7% via the oral route, and 100% subcutaneous route should be used to calculate criteria in health advisories. In assessing human exposure, best available data should be used to judge uptake and retention factors.

Such observations are relevant to the evaluation of risks from ingestion of contaminated soil or vegetation and/or inhalation of contaminated air in a site-specific situation.

More recently, ATSDR (1994) has published Minimal Risk Levels (MRL) in humans for "inorganic" mercury. An MRL is defined as an estimate of daily human exposure to a dose of a chemical that is likely to be without appreciable risk of adverse effects (noncancerous effects) over a specified period of exposure. An MRL of 0.007 mg Hg/kg/day (7 µg/kg/day) has been derived for acute oral exposure based on a NOAEL for renal effects in a 14-day rat study (NTP, 1993) and application of an uncertainty factor of 100.

An MRL of 0.002 mg/kg/day (2 µg/kg/day) was derived for intermediate exposure (6 months) to inorganic mercury (uncertainty factor = 100) based on the same study. No MRL for chronic exposure was derived because the study identifying the lowest LOAEL for chronic exposure observed a decreased survival rate for male rats at that dose level.

3.1.2 Inhalation

The U.S. EPA's Integrated Risk Information System database (IRIS, 1994) notes that the inhalation Reference Concentration (RfC) for inorganic mercury is currently under review and no value is available at this time.

In its 1992 edition of the Health Effects Assessment Summary Tables (HEAST), the U.S. EPA Office of Research and Development suggests an RfC for elemental mercury (Hg°) of 3×10^{-4} mg/m^3 (340 ng/m^3) set to prevent neurotoxic effects. It is noted, however, that this value is under review and, consequently, subject to change. The U.S. EPA defines an RfC as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

The WHO, in its 1987 Air Quality Guidelines for Europe, has not recommended any ambient air guideline based on the argument that absence of quantitative information on the consequences of the deposition of atmospheric mercury precludes the determination of the associated potential health effects (WHO, 1987). This same *status quo* seems to prevail today in the WHO policy on atmospheric mercury. However, WHO (1991) has derived a human **inhalation** exposure equivalent to the NOAEL for renal effects in animal studies as follows:

$$\textit{human inhalation equivalent} = \frac{0.0158\ \textit{mg/kg/day} \times 70\ \textit{kg} \times 100\%}{200\ \textit{m}^3}$$
$$= 0.019\ \textit{mg/kg/day}\ (1.38\ \textit{mg/day})$$
$$= 19\ \mu\textit{g/kg/day}\ (1380\ \mu\textit{g/kg/day})$$

It should be noted that the inhalation LOAEL calculated for kidney effects is well within the range of mercury vapour exposures in humans where

neurological and renal effects have been observed. This equivalent value does not incorporate traditional uncertainty factors in its derivation.

The ATSDR (1994) has recently derived inhalation MRLs for acute and chronic inhalation of metallic mercury vapours. The acute MRL is 2 x 10-5 mg/m^3 (25 ng/m^3) based on animal studies of changes in locomotor activity at 4 months of age and maze-solving speed. This value incorporates an uncertainty factor of 100. The chronic MRL of 1.4 x 10-5 mg/m^3 (14 ng/m^3) is based on studies of mercury-exposed workers in three industries. Adjustments were made for worker to lifetime exposure extrapolation and, in addition, an overall uncertainty factor of 100 was incorporated.

3.1.3 Dermal

No specific toxicological quantitative criteria for dermal exposure to mercury were located. Localized dermal effects are generally seen at very high concentrations in occupational settings or at levels formally present in various medicinal and cosmetic formulations. The environmental levels of mercury are very much lower than levels associated with these effects. No case reports of general environmental or soil exposures resulting in effects on the skin were found.

3.2 Methylmercury

It is currently under significant scientific debate as to whether or not a threshold for exposure to methylmercury exists below which no toxicological effects occur. In other words, similar to lead, there may be some small amount of neurological risk even with very small exposures to methylmercury. However, recent evidence reported in 1994 at the 12th International Neurotoxicology Conference regarding the Seychelles Child Development Study provided some suggestion that a No-Observed Effect Level (NOEL) may eventually be identifiable, and that this NOEL may be at higher mercury levels than those previously regarded as "threshold" levels. However, the children in these studies will have to be followed for several more years. There is therefore some pre-existing uncertainty regarding the underlying biological assumptions when assessing methylmercury exposure risk.

3.2.1 Ingestion

The IRIS database reports an oral RfD of 0.3 µg/kg-day for methylmercury based on a Low Observed Adverse Effect Level (LOAEL) for central nervous system effects of 3 µg/kg-day in humans (Uncertainty Factor = 10) (IRIS, 1994). A similar value is reported in the HEAST report (U.S. EPA, 1992). It is also noted that this value may change in the near future.

The California Air Pollution Control Officers Association, as reported in their California Air Toxics "Hot Spots" Program, Risk Assessment Guidelines (CAPCOA, 1992), has proposed an oral Acceptable Exposure Level (AEL) of 0.3 µg/kg/day, based on the 1991 U.S. EPA HEAST report. The AEL is used for the evaluation of potential non-cancer adverse health effects associated with long-term (chronic) exposures.

The WHO (1976) provided guidance on the establishment of long-term daily intake for mercury as methylmercury associated with the earliest effects in the most sensitive group in the adult population. At 3-7 µg/kg/day levels of consumption of methylmercury, it is expected that approximately 5% of the population will show early signs of methylmercury poisoning (parathesia). These levels are calculated for adults and there is sufficient reason to believe that they should be lower for children and pregnant women, due to methylmercury's ability to cross the placenta and because children are believed to be more susceptible to methylmercury's effects.

Cox and co-workers (1989), analyzing data from 83 infant-mother pairs from the outbreak of methylmercury poisoning in rural Iraq in 1971-1972, developed a novel dose-response analysis of infants prenatally exposed to methylmercury. Several thousand people with severe poisoning were admitted to local hospitals throughout the country. Both sexes and all age groups were affected. The Cox *et al.* (1989) analysis is based on application of a single-compartment model to single-strand hair analysis. The growth of human head hair recapitulates the blood concentration of mercury during the month that the hair segment is formed. Because human head hair grows about 1 cm per month, a 9 cm segment collected at the time of delivery of the infant would recapitulate the blood mercury concentration throughout the entire period of pregnancy (Amin-Zaki *et al.*, 1976). The results of this extensive maternal hair analysis were used to construct estimates of fetal exposure, and were examined against two measures of subsequent toxic effect in children examined at approximately 2-4 years of age. The investigators employed a logit and hockey stick models which they

and others have used previously in similar studies. Unlike conventional logit models, the hockey stick model assumes a population threshold for the response. Both models have a slow parameter rate of spontaneous response indicative of background. Inclusion of a background rate is a necessary part of any model for observational data such as is the case in this study. For both of the neurological endpoints considered by this study, retarded walking and CNS symptoms, a spontaneous level of response is quite likely. The fits of the logit and hockey stick models to the data on retarded walking and CNS symptoms are shown in Figures 3.1 and 3.2 together with the corresponding non-parametric confidence limits. The two figures show that parametric models are consistent with the data and with each other.

The maximum hair concentration during pregnancy appears to be a predictor of the risk of adverse developmental effects on the offspring. Independently of the type of statistical model used, a significant dose-response relationship is seen between maximum mercury concentrations in maternal hair and the frequency of abnormal development, whether this is indicated as a delay in walking or as abnormal neurological signs. As Cox *et al.* (1989) point out, traditionally, systemic mercury poisoning including prenatal poisoning has been treated as a threshold response with a definite threshold existing for the population.

The analysis suggested that using either model, a smooth relationship of increasing risk or response was presented with increasing maternal hair concentrations. It has previously been estimated that at adult hair levels of 50 ppm, the risk of the mildest form of poisoning is about 5% (WHO, 1976), whereas the Cox *et al.* (1989) best estimate for prenatal exposure is about 10-20%. Further the authors argue that the effect on children, i.e., psychomotor retardation is more serious than the mild sensory deficits which are seen in adults.

Cox *et al.* (1989) utilize the hockey stick model in order to estimate a threshold in terms of the estimated lowest effect level (ELEL), previously termed a practical threshold (Bakir *et al.* 1973), which corresponds to the point of intersection of the two straight lines on the hockey stick plots. These estimates of threshold depend on the assumed background of frequency. In the case of motor retardation, the background frequency is 0% and the corresponding ELEL is 7.3 ppm. If the upper limit of 4% background frequency is utilized, the value of the ELEL raises to 9 ppm. For central

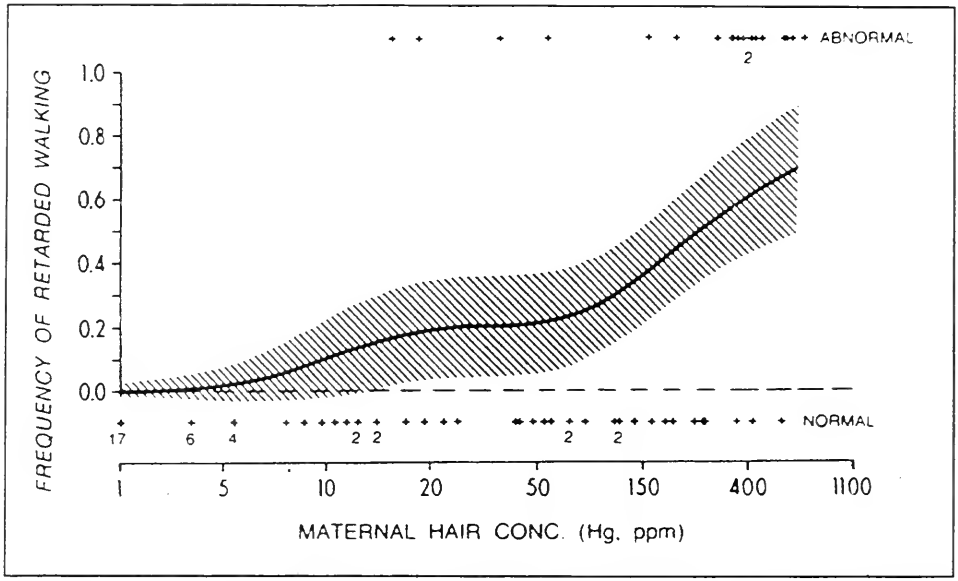

Figure 3.1: Non-parametric kernel smoothing analysis of the relationship between maximal maternal hair concentration of methyl mercury during pregnancy and retarded walking in offspring.

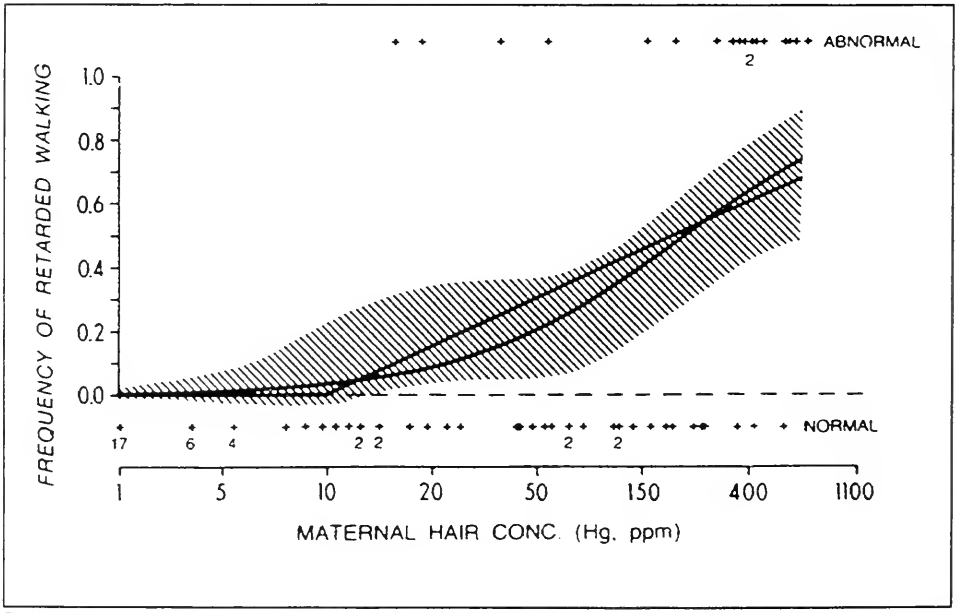

Figure 3.2: Logit and "hockey-stick" parametric models of dose-response between retarded walking and maximum maternal hair concentrations during pregnancy.

nervous system signs, the best estimate background is 9% and the corresponding ELEL is 10 ppm.

In summary, the Cox *et al.* (1989) analysis has suggested that motor retardation should occur in children prenatally exposed to maternal hair concentrations of less than 50 ppm and may be expected in the range of 10-20 ppm. Assuming the existence of a population threshold, the statistical best estimate places the value at about 10 ppm, but the 95% range of uncertainty includes 0-13.6 ppm. The WHO (1990), in assessing the health effects of methylmercury, has concluded that by considering the emerging model of neurological effects of methylmercury as a continuous dose-response relationship together with the extrapolation method of Cox *et al.* (1989), and the evaluation of other data quotations, it can be calculated that a maternal hair mercury concentration of 10-20 µg/g implies a 5% risk. The possibility cannot be excluded that effects detectable by psychological and behavioural testing or subclinical effects might occur at even lower levels of exposure, but evidence is currently lacking.

The fetus is at particular risk. Recent evidence shows that at a peak maternal hair mercury level above 70 ppm, there is a high risk (more than 30%) of neurological disorder in the offspring. A prudent interpretation of the Iraqi data implies that "a 5% risk may be associated with a peak mercury level of 10-20 micrograms/gram in the maternal hair" (WHO, 1990).

A critical question is the actual exposure level (or body burden) of methylmercury in humans which can lead to subtle changes in the offspring. The actual exposure levels and patterns are usually unknown, but the effect can be related to the hair mercury level and an approximate daily exposure calculated from the known kinetic parameters for methylmercury accumulation, distribution and excretion.

The WHO (1990) has determined that a daily methylmercury consumption of 0.48 µg/kg body weight will not result in any detectable adverse effect. However, daily intake of 3-7 µg/kg body weight would cause adverse effects on the nervous system manifested as an approximate 5% increase in the incidence of paraesthesia. Hair concentrations would be approximately 50-125 ppm at this level of intake. Clinical observations in the Iraqi study suggest that women are more sensitive to the toxic effect of methylmercury during pregnancy and therefore, they may be considered the most sensitive adult receptor.

ATSDR (1994) has derived an acute/intermediate MRL for methylmercury of 1.2 X 10-4 mg/kg/day or 0.12 µg/kg/day based on the delayed walking observations of Cox *et al.* (1989) and the statistical model of a population threshold. The lowest observed peak hair concentrations (14 ppm) associated with delayed onset of walking was converted to an equivalent blood concentration based on the assumption that hair concentration is 250 times blood concentration.

3.2.2 Inhalation

No inhalation reference concentration on the U.S. EPA IRIS database for organic mercury (as methylmercury) is available at this time (IRIS, 1994).

3.3 Total Mercury

Earlier exposure limits for mercury have often been expressed as total mercury partially because of the lack of species specific dose-response data and recognition of mercury exposure from all species needing to be considered. The most significant total mercury guideline for dietary consumption is that of the Joint FAO/WHO Expert Committee on Food Additives (FAO/WHO, 1972) which suggests a provisional tolerable weekly intake of 0.3 mg of total mercury per person of which no more than 0.2 mg should be present as methylmercury. This roughly equates to 42.8 µg/day over a week or 4.2 µg/kg/week for adults and 22.4 µg/kg/week for a child. A caveat on the WHO value is that: "If excessive intake is attributable entirely to inorganic mercury, the above provisional limit no longer applies and will need to be reassessed in light of all prevailing circumstances" (WHO, 1976).

The California Air Pollution Control Officers Association, as reported in their California Air Toxics "Hot Spots" Program, Risk Assessment Guidelines, has proposed for total mercury, an inhalation chronic AEL (Acceptable Exposure Level) of 3×10^{-4} mg/m^3, based on the 1991 U.S. EPA HEAST report (CAPCOA, 1992). The AEL is used for the evaluation of the potential noncancer adverse health effects of long-term (chronic) exposures.

3.4 Regulatory criteria

There are a large number of various regulatory guidelines and standards for mercury. In large part, these guidelines, while having a toxicological basis, may also be modified by a variety of technical and cost considerations. As such, they are not always the most appropriate criteria against which to assess risk but do give some basis for comparison regarding what is currently allowable in various jurisdictions. A comprehensive listing of these guidelines and their basis is provided in Appendix C.

Of particular note to the question of soil contamination in this community are the new MOEE guidelines for soil decommissioning and site clean-up. These are levels to which soil must be cleaned up to during a change of land use. The new value for mercury in residential, commercial and agricultural soils is 10 ppm. This is a health-based guideline based on the U.S. EPA chronic RfD of 3 X 10-4 mg/kg/day and various soil pathway modelling assumptions under the State of Massachusetts. It does not however take into account backyard vegetable consumption or soil-air vaporization of mercury.

4.0 HUMAN EXPOSURE ASSESSMENT

4.1 Overview of exposures from other chloralkali facilities and industrial sources of mercury

Atmospheric emissions from chloralkali facilities contribute to mercury pollution in the vicinity of these plants. This is demonstrated by mercury fallout pattern on snow (Jernelöv and Wallin, 1973) and accumulation of mercury by various biomonitoring plant species (Lodenius and Tulisalo, 1984; Calasans *et al.*, 1993).

A study which examined mercury concentrations in soil and biota collected near a chloralkali plant in Britain found higher concentrations of mercury in soil samples, grass (*Festuca rubra*) and earthworms (*Lumbricus terrestris*) collected closest to the plant (Bull *et al.*, 1977). Further, the authors of the study noted that tissue concentrations of mercury were higher in woodmice (*Apodemus sylvaticus*) and bank voles (*Clethrionomys glareolus*) collected in the vicinity of the chloralkali plant than those collected from uncontaminated rural control sites, suggesting that small mammals living in an area subjected to high mercury emissions can accumulate some of this mercury in their tissues.

Mercury levels were examined in air, grass and tissues of sheep grazing near a chloralkali facility in Britain (Edwards and Pumphrey, 1982). The average mercury concentration in air at the site was 0.14 $\mu g/m^3$. The mean concentration of mercury in the grass (dry weight) was 6.5 mg/kg for winter months and 1.9 mg/kg for summer months. Mercury was also detected in tissues of sheep although these levels were, according to the authors, in the range present in other commercially available meat. Tissues with the highest levels of mercury were the liver (0.6 mg/kg) and kidney (1.3 mg/kg). Less than 10% of the mercury was found in methylated form.

Soil and plant samples collected from sites in the area surrounding a chloralkali facility in India were found to have high levels of mercury (Shaw and Panigrahi, 1986). Leaves were found to have the highest residual mercury levels of all the different plant tissues examined, suggesting that grazing animals may be at risk of bioaccumulating mercury. Samples of muscle and liver tissue from goats and sheep collected from the local butcher shop were found to contain high levels of mercury. Mean mercury levels (on a wet weight bases were) were reported in goat liver and muscle as 51.51 ± 16.02 mg/kg and 2.86 ± 0.96 mg/kg, respectively. Mean mercury

levels in sheep liver and muscle tissue were reported as 46.26 ± 11.89 mg/kg and 2.91 ± 0.86 mg/kg, respectively. The authors surmised that human consumption of local meat could reasonably exceed the tolerable weekly intake of 0.3 mg total mercury established by the FAO/WHO Expert Committee on Food Additives in 1972. However, the mean blood mercury level of 18 human subjects living in the area was found to be 38.21 ng ± 0.89 ng/Ml, well below the level of possible toxicity.

Construction workers exposed to high levels of mercury during scheduled maintenance of a chloralkali plant in Tennessee in 1988 exhibited symptoms of acute mercury toxicity and chronic neurological disturbances (Bluhm *et al.*, 1992). Workers were not decontaminated prior to leaving the site, likely resulting in contamination of their homes. Concern regarding possible mercury exposure to family members of exposed workers prompted an investigation (ATSDR, 1990). The mean air level of mercury determined by 12-hour sampling in the homes of these families was 0.92 ± 0.85 $\mu g/m^3$. Levels of mercury in urine samples collected from household members of the exposed workers were within the reference range for the general population and were not indicative of possible mercury toxicity. The mean value of urinary mercury was 5.1 ± 4.4 ng/mL.

Children of mercury-exposed thermometer plant workers had significantly higher mercury levels in their urine compared to children from the same community whose parents were not employed by the thermometer plant (Hudson *et al.*, 1987). Analysis of air levels in homes revealed higher mercury concentrations in workers' home versus nonworkers' homes. A significant correlation was found between the urine mercury levels of the children and urine mercury levels of their exposed parents. The median urine mercury level in the workers' children was 25 µg/L. None of the children exhibited frank mercury toxicity or evidence of neurologic toxicity.

4.2 Multimedia exposure estimates in the Cornwall community

4.2.1 Forms of mercury in soil and vegetation

Certain species of soil bacteria are capable of reducing mercuric compounds intracellularly to produce Hg^0 which is released to the soil. The rate of mercury reduction measured in the laboratory is quite rapid by pure cultures of mercury reducers. However, population densities of bacteria capable of reducing mercury are extremely low in the natural soil environment and the

respective release of volatile mercury would not be appreciable enough to warrant consideration as a source of elemental mercury (Belliveau and Trevors, 1989).

Methylmercury is a highly lipophilic and highly neurotoxic form of mercury. Methylation of mercury is a biological process that occurs in anaerobic sediments and consequently does not apply to urban soils (Belliveau and Trevors, 1989). The emissions from the ICI facility are in the form of elemental mercury which is deposited largely through vapour fallout but also as particulate to some degree. In soil, this mercury will be found predominately as inorganic forms and mercuric salts or organocomplexes.

4.2.2 Exposures via ingestion

4.2.2.1 Ingestion of soil

Infants and young children ingest soil as a result of normal behavioural characteristics such as hand-to-mouth activity (i.e. finger licking and thumb sucking) and immature dietary habits (e.g. eating foods that have had direct contact with soil or dusts). The extent of such behaviours will vary between children and with age and therefore the amount of soil ingested will vary. Adults may also unwittingly ingest soil particles which have adhered to hands or food items.

4.2.2.1.1 Soil survey data

i) Phytotoxicology Surface Soil Survey data, 1991

Surface soil (0-5 cm) in the vicinity of the ICI facility in Cornwall was last surveyed in May and August of 1991, and previously sampled in 1976, 1978 and 1985. The results of these monitoring efforts can be found, along with the results of tree foliage and moss bag surveys, in the MOEE report entitled "*Phytotoxicology Assessment Survey in the Vicinity of ICI Forest Products and Domtar, Cornwall, 1987-1991*" (Dixon and Emerson, 1994).

The survey area was within approximately 3.5 km to the east and 1.7 km to the west of ICI, and comprised 22 sampling stations, numbered inconsecutively 1 through 37. All stations were south of the CNR line except station number 37. A map of the survey area illustrating the location of the

sampling stations can be found in Figure 4.1. Figure 4.1 also presents concentration contours calculated using the SURFER version 4.0 graphics package (see Appendix D for additional information). These contours are statistical approximations used to provide information on the approximate areas and/or patterns of contamination.

Table 4.1 presents the average of duplicate soil samples collected from individual sampling stations. The mean mercury concentration in surface soil samples collected in the vicinity of the ICI plant was 0.45 µg/g in May 1991, and 0.50 µg/g in August 1991.

Samples collected from stations 1, 2, 4, 6, 14, and 32 (May and August) and station number 21 (August only) had mercury concentrations which exceeded the Upper Limit of Normal (ULN) (see Appendix A) for urban Ontario soils. All the stations exceeding the ULN (with the exception of station 32) are west of Cumberland Avenue. High mercury concentrations in soil sampled at station 32, which is 1880 m away from the ICI mercury cell room, cannot be explained at this time and do not seem to be attributable to ICI facility. Station 1 is located west of Brookdale Avenue. Stations 2, 4, and 6 are east of Brookdale Avenue, and located closest to the residents of the community in the vicinity of Gulf and Yates Streets. The mean mercury concentration in rural control soil samples was found to be 0.05 in May 1991 and 0.04 in August 1991.

ii) Residential backyard garden soil study data, 1993

In 1993, the Phytotoxicology Section of the Standards Development Branch, MOEE carried out a survey of select backyard gardens to determine the mercury concentrations in homegrown vegetable produce and garden soil (Emerson, 1994). Seven sites were selected in the residential area immediately east of ICI. Garden site 8 was located on Edythe Avenue, west of the ICI facility. Two distant gardens were selected as control sites. Location of garden sites in close proximity to ICI is shown in Figure 4.2.

Mercury levels in garden soil samples are shown in Table 4.2. High mercury levels in surface soil were found at sites 1, 2, 3, 4, 5, and 6 (levels ranging from 0.19 to 2.37 µg/g) in comparison to control garden soil samples (levels ranging from 0.06 to 0.08 µg/g). Garden sites 1, 3, 5 and 6 had mercury concentrations which exceeded the ULN for urban soil of 0.05 µg/g.

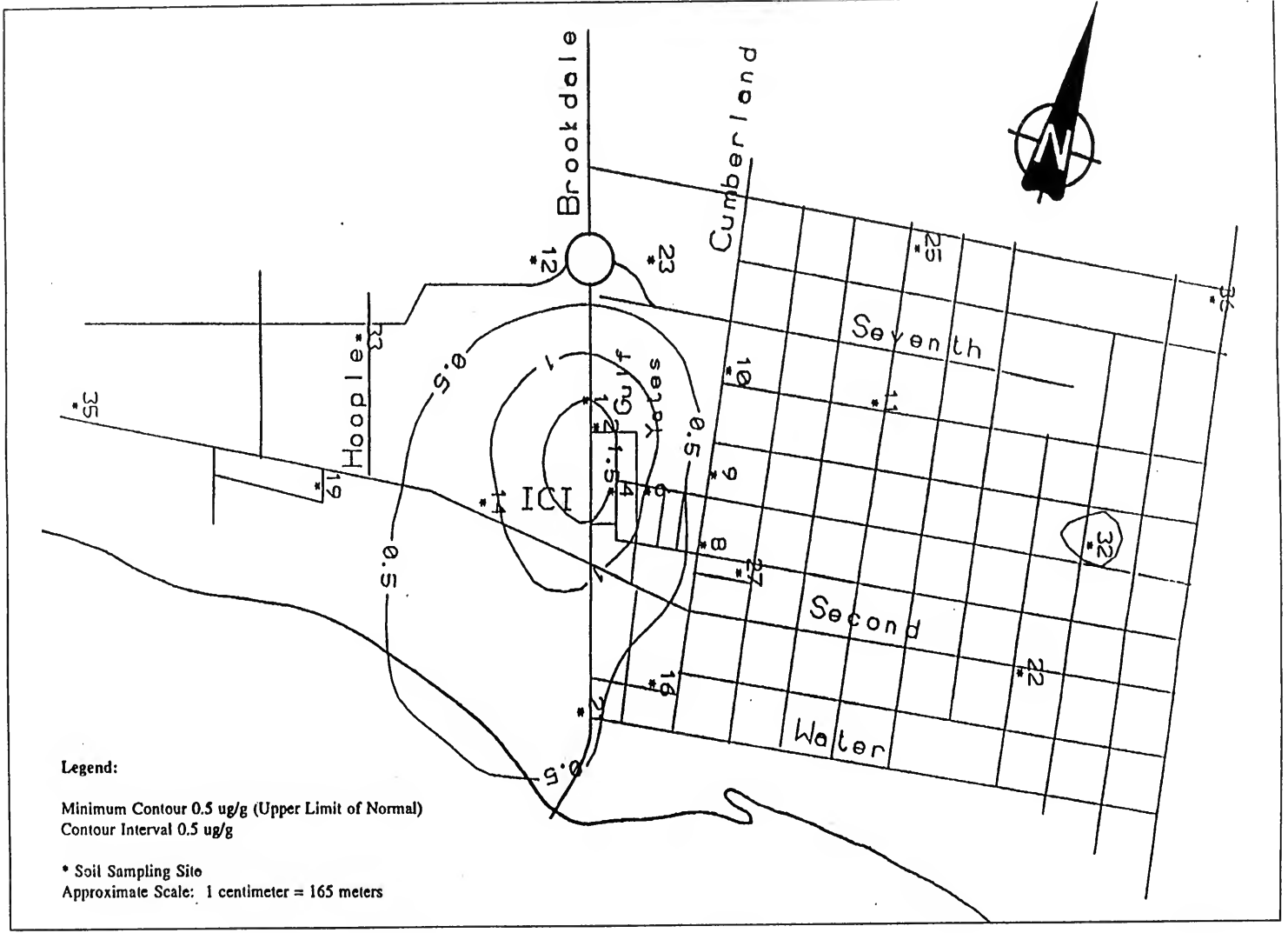

Figure 4.1: Mercury in surface soil: Cornwall (1991)

Table 4.1: Mercury concentration in surface soil sampled in the vicinity of ICI, Cornwall (1991)

Station #	Distance (m) and Direction from Hg Cell Room	Mercury Concentration in Surface Soil - 0-5 cm depth (µg/g, dry weight)[1]	
		May	August
1	360 N	1.07	1.55
2	360 NNE	1.70	1.85
4	240 E	1.75	1.95
6	100 ENE	0.84	0.81
8	560 E	0.39	0.38
9	600 ENE	0.36	0.27
10	750 NNE	0.29	0.27
11	1120 NE	0.39	0.43
12	680 NW	0.06	0.04
14	240 SW	0.60	0.83
16	760 ESE	0.18	0.17
19	760 SW	0.23	0.23
21	720 SSE	0.48	0.67
22	1700 E	0.18	0.15
23	760 N	0.07	0.06
25	1400 NNE	0.14	0.20
27	840 E	0.22	0.22
32	1880 ENE	0.60	0.57
33	700 W	0.11	0.08
35	1740 WSW	0.11	0.08
36	2300 NW	0.07	0.06
37	3500 NE	0.07	0.05
	Mean	**0.45**	**0.50**
	Minimum	**0.06**	**0.04**
	Maximum	**1.75**	**1.95**

[1] Mean of duplicate samples.

Note 1: Shaded soil concentrations exceed the ULN for urban Ontario soil (0.5 µg/g).

Dixon and Emerson, 1994

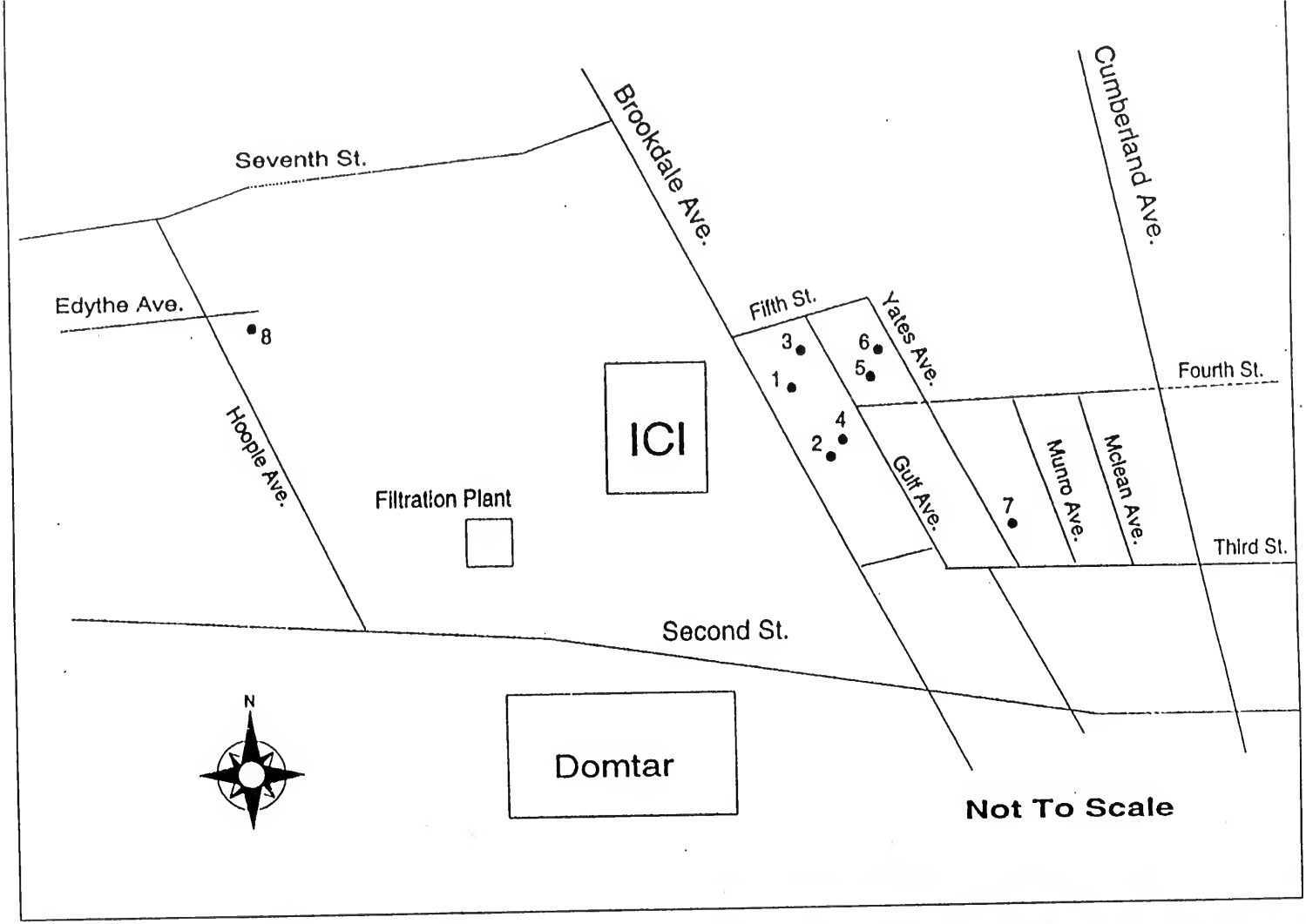

Figure 4.2: Sketch map showing approximate locations of garden sites in the immediate area of ICI (1993).

Table 4.2: **Mercury Concentrations in backyard garden soil, Cornwall (1993).**

Site #	Garden Location	Mercury Concentration in Surface Soil - 0-15 cm depth (µg/g, dry weight)	
		Jun.1[a]	Aug.18[b]
Gardens Close to ICI			
1	Brookdale Ave.	2.30	2.37
2	Brookdale Ave.	0.19	0.20
3	Gulf St.	1.20	1.63
4	Gulf St.	0.22	0.26
5	Gulf St.	2.20	2.10
6	Yates Ave.	0.49	0.59
7	Munro Ave.	0.07	0.07
8	Edythe Ave.	0.05 T	0.08
Mean		**0.84**	**0.91**
Minimum		**0.05**	**0.07**
Maximum		**2.30**	**2.37**
Gardens Distant from ICI			
9	Eighth St.	0.06	0.08
10	Grant Ave.	0.07	0.06
Mean		**0.07**	**0.07**
Minimum		**0.06**	**0.06**
Maximum		**0.07**	**0.08**

[a] Denotes the mean of duplicate samples
[b] Denotes the mean of triplicate samples
T A measurable trace amount: interpret with caution.
Note 1: Shaded soil concentrations exceed urban ULN guideline of 0.5 µg/g.
Emerson, 1994

iii) Phytotoxicology Soil Surveys - Historical Data

As indicated previously, surface soil in the vicinity of ICI was sampled previously in 1976, 1978, and 1985 (Dixon and Emerson, 1994). The mean and range of mercury concentrations in soil samples collected from sampling stations in the vicinity of the ICI facility in all MOEE survey years is presented in Table 4.3. Data can be found in Appendix E.

Table 4.3: Summary of mercury concentrations (µg/g) in surface soil samples (0-5 cm) collected in the vicinity of ICI, Cornwall

Survey Year	Concentration (µg/g)*		% stations exceeding urban ULN
	Mean	Range	
1976	0.59	0.05 - 2.60	28
1978	0.81	0.02 - 4.30	32
1985	0.77	0.06 - 3.70	32
1991 (May)	0.45	0.06 - 1.75	27
1991 (August)	0.50	0.04 - 1.95	32

* Mean of triplicate samples in 1976 and 1978, and mean of duplicate samples in 1985 and 1991.

Dixon and Emerson, 1994.

In all MOEE survey years (1976, 1977, 1985, 1991), stations exceeding the ULN were within 1000 m of the ICI facility, with the exception of station #32 which exceeded the ULN in both May and August 1991.

The percentage of stations exceeding the urban ULN guideline was 27% in May 1991 and 32% in August 1991. This is consistent with previous survey years: 28%, 32%, and 32% exceedence in 1976, 1978 and 1985, respectively. Although the mean mercury concentration in the soil (averaged from all sampling stations) has steadily decreased, stations closest to the ICI facility continue to exceed the ULN. Decline in mean mercury concentration in soil over the entire survey area is consistent with the overall declining trend in mercury releases to the atmosphere from the ICI plant over the last 15 years.

4.2.2.1.2 Estimate of mercury exposure from ingestion of soil

The mean concentration of mercury calculated from data obtained from the August 1991 survey was used in the determination of mercury intake from Cornwall-area soil as this represents the most extensive survey area (22 sites located throughout the affected community) and is also fairly recent. Data from the August 1991 survey was used over the May 1991 data as the mean mercury concentration is somewhat higher, resulting in a more conservative exposure estimate and because this data set provides a wider range of mercury concentrations (0.04 - 1.95 µg/g). However, higher soil mercury concentrations were seen in the 1993 backyard garden survey. As such, exposures were also calculated using the maximum soil mercury concentration from this survey.

The estimated intake of mercury from ingestion of soil is calculated by solving for the following equation:

$$Intake = C_{soil} \times IR$$

where,

C_{soil} = concentration of mercury in soil
IR = contact rate/ingestion rate

The available scientific information concerning soil ingestion rates is summarized in Appendix F. Taking into account the available information, a value of 80 mg/day was selected as the soil intake rate for a child and 20 mg/day was selected as the soil intake rate for adults. These assumptions are in keeping with the recommended Canadian Reference values for dirt, dust and soil intake (HWC, 1988).

A summary of the estimated intake of mercury from soil is presented in Table 4.4. The average estimated intake for children (0.5 months-4 years) and adults (>20 years) are 0.04 and 0.01 µg/day, respectively. The maximum value, site 1 from the backyard garden survey results in an estimated intake of 0.19 µg/day for children and 0.05 µg/day for adults, greater than the mean by approximately 20%. Intake estimates were also calculated using the modelled concentration values.

Soil exposures are based on continual daily exposure. They are not adjusted for factors such as climate, time spent indoors and plausible

residence time in the community. Adjustment for any or all of these factors would lower exposure estimates. Therefore, this model will tend to overestimate exposure.

Soil exposures are expressed as intake values where compared to reference doses, which are based on administered dose and not adjusted for absorption rates. Little information is available regarding the bioavailability of mercury from soil.

Table 4.4: Estimated intake of mercury from soil

Mercury concentration in soil (µg/g)		Estimated intake of mercury from soil			
		Child (0.5-4 years)[1]		Adult (>20 years)[2]	
		µg/day	µg/kg/day	µg/day	µg/kg/day
Soil Survey Data					
Mean[3]	0.50	0.04	0.003	0.01	0.0001
Minimum[3]	0.04	0.003	0.0002	0.001	0.00001
Maximum[4]	2.37	0.19	0.01	0.05	0.0007
Modelled Concentrations using SURFER					
Contour 1	0.5	0.04	0.003	0.01	0.0001
Contour 2	1	0.08	0.006	0.02	0.0003
Contour 3	1.5	0.12	0.009	0.03	0.0004

[1] Based on a 13.3. kg child and an average soil ingestion rate of 0.08 g/day.
[2] Based on a 70 kg adult and an average soil ingestion rate of 0.02 g/day.
[3] Mean and minimum values taken from Phytotoxicology Surface Soil survey, 1991.
[4] Maximum value from backyard garden survey (site 1, sampled August 18, 1993).

4.2.2.2 Ingestion of backyard garden vegetables

Those individuals who grow and consume vegetables grown on residential properties may have additional mercury intakes by this exposure pathway.

In order to examine the question of intake from homegrown produce, concentrations of contaminants in produce are combined with various models regarding adult and child consumption of these foodstuffs to yield

crude estimates of intake. These are utilized to provide (1) an indication of to what degree certain individuals may have increased mercury exposures if consuming homegrown produce and (2) for gross comparison to other exposure pathway intakes.

4.2.2.2.1 Backyard garden vegetable survey data

As indicated in section 4.2.2.1.1 (ii), the MOEE undertook a survey of select backyard gardens to determine the mercury concentrations in edible vegetable produce and garden soil in the vicinity of ICI (Emerson, 1994).

In early June 1993, three common vegetable crops (leaf lettuce, beets and tomatoes) were established in 8 existing gardens close to ICI and two distant control gardens (east and west of ICI). Location of garden sites is detailed in section 4.2.2.1.1 and illustrated in Figure 4.2. Seeds and transplants were planted in the first week of June and maintained by the garden owner as part of their own vegetable garden.

The leaf lettuce was collected in July and again in August (except at site 1, 6 and 10 where only one collection was possible). Beets and tomatoes were collected once in August. Where possible, the sampling was done in triplicate. In cases where there was insufficient material, single or duplicate samples were collected.

Vegetable samples were processed (washed, weighed, dried, weighed again and ground) prior to submission for mercury analysis to the MOEE Laboratory Services Branch. The vegetable samples were weighed on a fresh and dry weight basis to determine the percent moisture loss from drying so that the reported dry weight concentrations could be converted back to fresh weight (as consumed) values. The analysis of mercury was done by cold vapour atomic absorption spectrophotometry. This analytical procedure has a detection limit of 0.01 µg/g, dry weight (D. Russell, 1994, personal communication).

Background levels of mercury in plant tissue can range from 0.03 to 0.07 ppm, dry weight (Kuja, 1991). The concentrations of mercury in backyard garden vegetables sampled in the vicinity of the ICI facility in 1993 is presented in Table 4.5. Vegetables sampled from the control sites, #9 and #10, were within the background range (less than detection to 0.06 ppm). The highest mercury concentrations where seen in vegetables sampled from

garden sites located close to ICI: these ranged from 0.03 to 1.3 ppm for lettuce, 0.10 to 0.89 ppm for beet tops, and less than detection to 0.02 ppm for both beet roots and tomato fruits.

Higher mercury levels were found in garden vegetables sampled from sites 1, 2, 3, 4, and 5. These sites are all east of the ICI facility and west of Yates Avenue. Conversion to fresh weight mercury concentrations revealed that beet tops at these sites exceeded the former World Health Organization (WHO) guideline for mercury of 0.05 μg/g for vegetable crops. Mercury concentrations in leaf lettuce and beet tops grown in gardens in the vicinity of ICI Forest Products were both well above normal.

4.2.2.2.2 Estimates of mercury exposure from ingestion of backyard garden vegetables

The amounts and types of produce that people might consume from a backyard garden are influenced by the size of the garden, the yields of the crops grown, and the preferences of the individuals.

The dose of mercury received from consumption of backyard garden vegetables is calculated using the three vegetable consumption models described below. Time-averaged exposure values are required to evaluate chronic toxicity. Incidental exposure estimates are required for consideration of acute effects and comparison with acceptable daily intakes.

1. **Low-Level Chronic Exposure:** The vegetables sampled are representative of a varied garden produce which is consumed at a rate which is averaged over a year. This model considers that only a certain fraction of fruits and vegetables will be provided by the garden over the course of the year and assumes freezing of some vegetables for later consumption. Nutrition Canada Survey data from 1972 for Ontario indicates that the average daily consumption of fruits, fruit products and vegetables combined is 372 g/day (26% of total diet) for children one to four years old, and 489 g/day (32% of total diet) for adult males (NCS, 1972). According to the produce module of the AERIS Model (Aid for Evaluating the Redevelopment of Industrial Sites) for soil-related exposures, the yields of various types of crops generally range from 0.3 to 2.6 kg/m^2. Backyard gardens, which generally consist of a mixture of crops, may yield approximately 1.4 kg/m^2, although it is possible to achieve higher yields with special

Table 4.5: Mercury concentrations in backyard garden vegetables and soil, Cornwall (1993).

Site No.	Garden Location	Mercury Concentration (µg/g)*						
		Surface Soil (0-15 cm depth)		Leaf Lettuce		Beet Tops	Beet Roots	Tomato Fruits
		Jun.1[b]	Aug.18	Jul.14	Aug.18	Aug.18	Aug.18	Aug.26
Gardens Close to ICI								
1	Brookdale Ave.	2.30	2.37	NR	0.68 (0.03)	0.89[a] (0.07)	NR	0.02[a]T (0.002)
2	Brookdale Ave.	0.19	0.20	1.30 (0.03)	0.67 (0.03)	0.84 (0.07)	0.02 T (0.002)	0.02 T (0.002)
3	Gulf St.	1.20	1.63	0.56 (0.01)	0.91 (0.04)	0.69 (0.06)	0.005 DL (0.0005)	0.005 DL (0.0004)
4	Gulf St.	0.22	0.26	1.30 (0.03)	0.81[a] (0.03)	0.84[b] (0.07)	0.005 DL[a] (0.0005)	0.02 T (0.002)
5	Gulf St.	2.20	2.10	0.64 (0.01)	0.46[a] (0.02)	0.76 (0.06)	0.005 DL (0.0005)	0.005 DL (0.0004)
6	Yates Ave.	0.49	0.59	0.35 (0.007)	NR	0.24[a] (0.02)	0.005 DL[a] (0.0005)	0.005 DL (0.0004)
7	Munro Ave.	0.07	0.07	0.43 (0.009)	0.16 (0.006)	0.25 (0.02)	0.005 DL[b] (0.0005)	0.005 DL (0.0004)
8	Edythe Ave.	0.05 T	0.08	0.05 T (0.001)	0.03 T (0.001)	0.10 (0.008)	0.005 DL (0.0005)	0.005 DL (0.0004)
Distant Gardens								
9	Eighth St.	0.06	0.08	0.06 (0.001)	0.04 T (0.002)	0.06 (0.005)	0.005 DL (0.0005)	0.005 DL (0.0004)
10	Grant Ave.	0.07	0.06	0.03 T (0.0006)	NR	0.03 T (0.002)	0.005 DL[a] (0.0005)	0.005 DL[a] (0.0004)

* Mean concentration of triplicate samples (with the exception of those with a subscript) expressed as µg/g, dry weight.

Numbers in brackets indicate mercury as fresh weight (as consumed) concentrations (To convert vegetable data to fresh weight levels, multiply dry weight levels by the following conversion factors: lettuce (July: 0.02; August: 0.04); beet tops (0.08); beet roots (0.1); tomato (0.08). These conversion factors were derived from the average percent moisture content of each vegetable crop (determined from fresh and dry weights) using the following formula: Conversion Factor = (100 - % moisture)/100.

[a] Indicates that there was only enough material for a single sample.

[b] Denotes the mean of duplicate samples. In June, all soil sites were sampled in duplicate.

NR No results. Samples not collected because of insufficient sample material.

DL At or below analytical detection limit of 0.01 µg/g. For these samples, half DL was substituted for dry weight mercury concentration.

T A measurable trace amount, interpret with caution.

Note 1: Shaded soil concentrations exceed urban ULN guideline of 0.5 µg/g.

Note 2: Shaded vegetable concentrations exceed the former WHO guideline of 0.05 µg/g, when converted to fresh weight concentration.

Emerson, 1994

techniques or extraordinary efforts. Assuming a garden area of 30 m^2, the total yield of produce from a backyard garden would be 42 kg. This represents 13% of the vegetables and fruits that one adult and one child would consume in one year. In a family of four, approximately 7% of fruits and vegetables consumed by each individual could be garden grown. Assuming the family consumes all the vegetables grown in their backyard garden, consumption of homegrown produce would account for 1.8% (**26 g/day**) of the diet of children and 2.2% (**34 g/day**) of the diet of resident adults, averaged over a year.

2. **Limited Low Level Chronic Exposure:** Only lettuce, beets and tomatoes are eaten. The vegetables sampled are consumed at the typical consumption rates, averaged over a year. Typical consumption rates/suggested mean daily intakes for lettuce, beets, and tomato fruits are shown in Table 4.6.

Table 4.6: Average Vegetable-Specific Food Consumption Rates

Vegetable	Consumption Rate (g/day)	
	Child	Adult
Lettuce	2.37	12.7
Beets[1]	0.43	1.8
Tomato Fruits	3.19	17.9

[1] This consumption rate was used for estimate of intake for both roots and tops.
Health Canada, 1992

3. **Acute exposure:** This scenario is provided to account for those days, particularly during the growing season, where all of the daily intake of vegetables is taken from the gardens. This assumes that the vegetables sampled are representative of what is eaten. Average Ontario intake for potatoes and other vegetables (excluding fruits) is approximately **98 g/day** for children and **326 g/day** for adult males.

The major difference of model 3 from the other models is that the exposure is not averaged over the entire year, but rather exposure results from consumption of all vegetables over a for short periods. Models 1 and 2 estimate chronic exposure, appropriate for comparison to dose-response information based on chronic exposure.

The estimated intakes of mercury from consumption of vegetables grown in the vicinity of the ICI facility is presented in Table 7.

The estimated human intake is given by the following:

$$Intake = C_{vegetables} \times IR$$

where,

$C_{vegetables}$ = concentration of mercury in vegetables (fresh weight, µg/g))

IR = ingestion rate (g/day)

The ingestion rate is based on the three models of vegetable consumption described in this section.

Food preparation practices such as washing, peeling and cooking, which have not been taken into account in the following estimates, will likely lower the actual ingested dose of mercury. Further, consumption rates for adults were based on those of adult males; adult female consumption rates may be somewhat lower.

Table 4.7: Estimated intake of mercury from ingestion of backyard vegetables

Site #	Vegetable	Fresh Weight (µg/g)	Estimated intake of mercury from backyard vegetables (µg/day)					
			Model 1[1]		Model 2[2]		Model 3[3]	
			Child	Adult	Child	Adult	Child	Adult
1	beet root	NR	-	-	-	-	-	-
	beet top	0.07	1.82	2.38	0.03	0.13	6.86	22.82
	lettuce-Aug.	0.030	0.78	1.02	0.07	0.38	2.94	9.78
	tomato	0.002	0.05	0.07	0.01	0.04	0.20	0.65
2	beet root	0.002	0.05	0.07	0.0009	0.004	0.20	0.65
	beet top	0.07	1.82	2.38	0.03	0.13	6.86	22.82
	lettuce[m]	0.03	0.78	1.02	0.07	0.38	2.94	9.78
	tomato	0.002	0.05	0.07	0.006	0.04	0.20	0.65
3	beet root	0.0005	0.01	0.02	0.0002	0.0009	0.05	0.16
	beet top	0.06	1.56	2.04	0.03	0.11	5.88	19.56
	lettuce[m]	0.03	0.78	1.02	0.07	0.38	2.94	9.78
	tomato	0.0004	0.01	0.01	0.001	0.007	0.04	0.13
4	beet root	0.0005	0.01	0.02	0.0002	0.0009	0.05	0.16
	beet top	0.07	1.82	2.38	0.03	0.13	6.86	22.82
	lettuce[m]	0.03	0.78	1.02	0.07	0.38	2.94	9.78
	tomato	0.002	0.05	0.07	0.01	0.04	0.20	0.65
5	beet root	0.0005	0.01	0.02	0.0002	0.0009	0.05	0.16
	beet top	0.06	1.56	2.04	0.03	0.11	5.88	19.56
	lettuce[m]	0.02	0.52	0.68	0.05	0.25	1.96	6.52
	tomato	0.0004	0.01	0.01	0.001	0.01	0.04	0.13
6	beet root	0.0005	0.01	0.02	0.0002	0.001	0.05	0.16
	beet top	0.02	0.52	0.68	0.01	0.04	1.96	6.52
	lettuce-Jul.	0.01	0.26	0.34	0.02	0.13	0.98	3.26
	tomato	0.0004	0.01	0.01	0.001	0.01	0.04	0.13
7	beet root	0.0005	0.01	0.02	0.0002	0.001	0.05	0.16
	beet top	0.02	0.52	0.68	0.009	0.04	1.96	6.52
	lettuce[m]	0.01	0.26	0.34	0.02	0.13	0.98	3.26
	tomato	0.0004	0.01	0.01	0.001	0.007	0.04	0.13

Table 4.7: **Estimated intake of mercury from ingestion of backyard vegetables**

Site #	Vegetable	Fresh Weight (µg/g)	Estimated intake of mercury from backyard vegetables (µg/day)					
			Model 1[1]		Model 2[2]		Model 3[3]	
			Child	Adult	Child	Adult	Child	Adult
8	beet root	0.0005	0.01	0.02	0.0002	0.0009	0.05	0.16
	beet top	0.008	0.21	0.27	0.003	0.01	0.78	2.61
	lettuce[m]	0.001	0.03	0.03	0.002	0.013	0.10	0.33
	tomato	0.0004	0.01	0.01	0.001	0.007	0.04	0.13
Mean		**0.020**	**0.52**	**0.68**			**1.96**	**6.52**
Minimum		**0.0004**	**0.01**	**0.01**			**0.04**	**0.13**
Maximum		**0.07**	**1.82**	**2.38**			**6.86**	**22.82**
9	beet root	0.0005	0.01	0.02	0.0002	0.0009	0.05	0.16
	beet top	0.005	0.13	0.17	0.002	0.009	0.49	1.63
	lettuce[m]	0.002	0.05	0.07	0.005	0.03	0.20	0.65
	tomato	0.0004	0.01	0.01	0.001	0.007	0.04	0.13
10	beet root	0.0005	0.01	0.02	0.0002	0.0009	0.05	0.16
	beet top	0.002	0.05	0.07	0.0009	0.004	0.20	0.65
	lettuce-Jul.	0.001	0.03	0.03	0.0024	0.01	0.10	0.33
	tomato	0.0004	0.01	0.01	0.001	0.007	0.04	0.13
Mean		**0.001**	**0.03**	**0.03**			**0.10**	**0.33**
Minimum		**0.0004**	**0.010**	**0.01**			**0.04**	**0.13**
Maximum		**0.005**	**0.13**	**0.17**			**0.49**	**1.63**

[1] Model 1: children 26 g/day; adults 34 g/day.
[2] Model 2: lettuce, beets, tomato fruits for children 2.37, 0.43, and 3.19 g/day, respectively and for adults 12.7, 1.8, and 17.9 g/day, respectively.
[3] Model 3: children 98/day; adults 326 g/day.
[m] fresh weight concentrations of mercury of lettuce sampled in July and August were averaged.

Table 4.8: **Summary of estimated intake of mercury from chronic consumption (Model 1) and acute consumption (Model 3) of backyard garden vegetables grown in gardens close to ICI**

Mercury concentration in backyard garden vegetables (µg/g - fresh weight)		Estimated intake of mercury from consumption of garden vegetables			
		Child (0.5-4 years)[1]		Adult (>20 years)[2]	
		µg/day	µg/kg/day	µg/day	µg/kg/day
Model 1[3]					
Mean	0.02	0.52	0.04	0.68	0.01
Minimum	0.0004	0.01	0.0008	0.01	0.0001
Maximum	0.07	1.82	0.14	2.38	0.03
Model 3[4]					
Mean	0.02	1.96	0.15	6.52	0.09
Minimum	0.0004	0.04	0.003	0.13	0.002
Maximum	0.07	6.86	0.52	22.82	0.33

[1] Based on a 13.3 kg child.
[2] Based on a 70 kg adult.
[3] Based on a vegetable consumption rate of 26 g/day for children and 34 g/day for adults.
[4] Based on a vegetable consumption rate of 98 g/day for children and 326 g/day for adults.

4.2.2.3 Ingestion of food (background)

4.2.2.3.1 Mercury concentrations in various foods

Because of its ubiquitous presence in the environment, mercury is found in minute amounts in all foods. Average amounts of total mercury present in various food groups are listed in Table 4.9. It is believed that most mercury present in food, except in special circumstances, is in the inorganic form. A significant portion of the mercury present in fish, however, is in the form of methylmercury.

Table 4.9: Average mercury concentrations present in various food groups

Food Group	Average total mercury concentration
Fruits	<0.01 - 0.02 ppm
Vegetables	<0.01 - 0.04 ppm
Potatoes	0.6 ppb
Dairy Products	<0.1 - 0.1 ppb
Cereal and grain products	0.9 - 2.1 ppb
Meat - muscle	<1.0 - 2.0 ppb
Meat - liver, kidney	<1.0 - 3.0 ppb
Fish and fish products	<0.01 - >1.0 ppm

Bob Hills, 1994, personal communication

4.2.2.3.2 Dietary exposure estimates for the general Canadian population

Based on their total diet study of 1982-1984, the FDA has estimated that the average intake rate for total mercury is between 50-100 ng/kg/day, with methylmercury contributing approximately 80-90% of this figure (Gunderson, 1988). A separate survey of fish consumers revealed an average intake of methylmercury to be 36 ng/kg/day, with a 99% upper bound at 243 ng/kg/day (Clarkson, 1990).

The average daily intake of mercury from food in Canada was estimated to be 0.02 mg/person in 1964, and 0.013 mg/person in 1974 (HWC, 1986). More recently, Mitchell (1992) estimated the average daily intake of mercury from food as 3.59 µg/day for children (0.5 - 4 years) and 9.40 µg/day for adults (≥ 20 years). In this case, dietary intake of mercury was estimated by multiplying the average daily intake estimates for food group composites by estimates of the mercury content of these composites. Speciation was assumed to be 20% inorganic, and 80% methylmercury for fish (and fish products) and 100% inorganic for non-fish foods. Table 4.10 outlines the estimated intake of inorganic mercury and methylmercury from food based on Mitchell's estimates.

Table 4.10: Estimated intake of mercury from food

Food Group	Estimated intake of mercury from consumption of food							
	Child (0.5-4 years)				Adult (>20 years)			
	Inorganic		Methyl		Inorganic		Methyl	
	µg/day	µg/kg/day	µg/day	µg/kg/day	µg/day	µg/kg/day	µg/day	µg/kg/day
Fish	0.07	0.005	0.28	0.02	0.28	0.004	1.10	0.02
Non-Fish	3.24	0.24	-	-	8.02	0.11	-	-

Mitchell, 1992

4.2.2.4 Ingestion of locally caught fish

4.2.2.4.1 Fish survey data

Fish consumption represents the largest non-occupational exposure to mercury. Further, much of the mercury present in fish is in the methylated form. As such, recreational anglers and their families, as well as other groups which consume greater than average amounts of fish (such as aboriginal communities), may be at additional risk of mercury toxicity. It is therefore important to have an accurate assessment of the mercury levels in Cornwall area fish and of the rate at which these fish are consumed.

The Ontario government monitors contaminant levels in sport fish through the Sport Fish Contaminant Monitoring Program. Table 4.11 presents data collected through this program. The levels of mercury contamination, weight and length of fish surveyed in 1989 in the St. Lawrence River - Cornwall Island area and surveyed in 1992 in Lake St. Francis are reported (MOEE, unpublished data).

For reference purposes, the current Canadian guideline for total mercury in fish is 0.5 ppm (Bob Hills, 1994, personal communication). This guideline was established in 1969 by Health Protection Branch of Health Canada.

Table 4.11: Mercury concentration, length and weight of fish surveyed through the Sport Fish Contaminant Monitoring Program

Species	Number of fish surveyed	Hg concentration (µg/g)[1] *Mean (min, max)*	Weight (g) *Mean (min, max)*	Length (cm) *Mean (min, max)*	Correlation between length and [Hg]
St. Lawrence River - Cornwall Island area (1989)					
Yellow Perch	18	0.33 (0.17, 0.54)	234 (126, 347)	22.9 (20.3, 26)	0.61
Northern Pike	14	0.59 (0.23, 1.4)	1157 (471, 3213)	48.4 (36, 70)	0.83
Walleye	5	0.46 (0.3, 0.55)	886 (287, 1942)	39.4 (31, 52.5)	0.80
White Sucker	1	0.73	1640	48	-
Redhorse Sucker	2	0.22 (0.1, 0.33)	1579 (1046, 2111)	41 (36, 46)	-
Carp	7	0.32 (0.1, 0.57)	6099 (723, 9842)	60 (30, 79)	0.96
Brown Bullhead	11	0.25 (0.11, 0.53)	309 (86, 473)	25.7 (22, 28)	0.75
Lake St. Francis (1992)					
Yellow Perch	31	0.19 (0.09, 0.32)	189 (80, 380)	23.9 (18.2, 31.5)	not done
Pike	25	0.60 (0.20, 1.30)	1708 (560, 4450)	63.3 (47.6, 89.2)	not done
Smallmouth Bass	13	0.40 (0.24, 0.59)	1162 (470, 1600)	39.9 (31.5, 46.0)	not done
Walleye	7	1.01 (0.48, 2.00)	2479 (1600, 3500)	62.4 (52.0, 75.0)	not done

[1] Reported concentrations are for total Hg.

MOEE, unpublished data

Figure 4.3 illustrates the trend of total mercury content in various fish caught in the area in 1978, 1986 and 1989. As can be see from this illustration, the mercury concentration will fluctuate within and between species from year to year. Northern Pike and Walleye have shown a steady decline in mercury contamination. In contrast, mercury levels in Yellow Perch were highest in 1989, although the concentrations are not as high as Northern Pike and Walleye. The mercury concentration in fish will depend, not only on the varying levels of mercury in the water body, but also on the varying length,

age and weight of the fish sampled. This may be influencing the reported mean concentrations of mercury. For example, in 1989, an increase in mean mercury concentrations was observed in yellow perch, over 1986 and 1978. However, in 1989, fish surveyed were longer on average, than those surveyed in previous years (data not shown), indicating older fish. Therefore, this marginal increase in mercury in yellow perch should be controlled for length and weight of fish. The results of a regression analysis comparing length with mercury concentration for the 1989 data resulting in correlations greater that 0.60 for all species (Table 4.11) indicating a very strong relationship between the two parameters. Therefore, the intraspecies difference in mercury levels can be explained partially by the differential in accumulation with varying age and size. The interspecies variation can be attributed to where the individual species lies in the food chain.

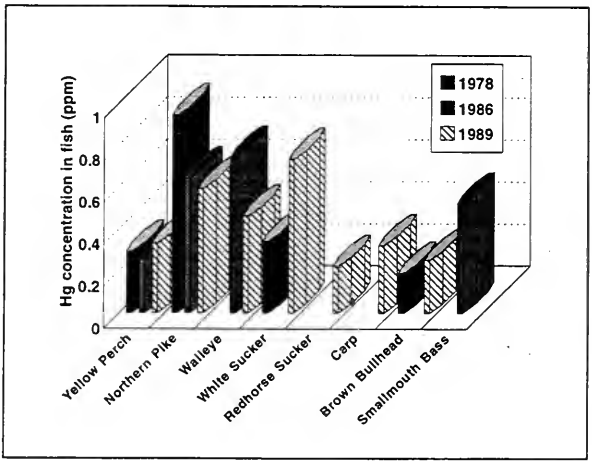

Figure 4.3: Mercury Concentrations in Fish in the St. Lawrence River (Cornwall Island): 1978-1989.

4.2.2.4.2 Estimate of mercury exposure from consumption of locally caught fish

In 1992, a questionnaire was included in the Guide to Eating Ontario Sport Fish to get a sense of the effectiveness of the consumption advice, fishing frequency, the most fished locations and the fish consumption patterns (Cox *et al.*, 1993). For the purposes of this exposure analysis, the fish consumption figure of 22.5 g/day derived from this questionnaire was used to estimate the exposure of adult anglers (see Appendix G for details of derivation). This figure represents the most recent data collected on fish consumption in the Cornwall region.

It is recognized that children will consume some angled fish, in particular where vacationing is associated with fishing activities. However, no

information on fish consumption patterns of children was included in the above mentioned questionnaire. Therefore, for the purposes of this exposure analysis, a daily fish consumption rate of 1.12 g/day was used to calculate mercury intake from angled sport fish. This intake rate is based on data from the 1970-72 Nutrition Canada Survey regarding consumption of cooked freshwater fish (NCS, 1972).

Table 4.12 presents the estimated intake of mercury from consumption of various fish species caught and eaten in Southern Ontario. The data from the 1992 Lake St. Francis fish survey was used as this represents a popular fishing spot in the Cornwall area and because this data is somewhat more recent. The mean mercury concentration of 0.55 µg/g is actually the mean of the species means. The minimum concentration represents the lowest mean (0.19, yellow perch) and the maximum concentration is the highest mean (1.01, walleye) from this 1992 survey.

Mercury in fish is assumed to be 80% methylmercury and 20% inorganic mercury. For adults, total mercury intake from this source, using the Lake St. Francis 1992 survey data, ranges from 0.06 - 0.32 µg/kg/day with a mean of 0.18 µg/kg/day. For children, total mercury intake from this source ranges from 0.013 - 0.09 µg/kg/day, averaging 0.049 µg/kg/day.

Table 4.12: Estimated intake of mercury from angled fish

Mercury concentration in angled fish (µg/g)[1]		Estimated intake of mercury from angled fish							
		Child (0.5 - 4 years)[2]				Adult (> 20 years)[3]			
		Inorganic		Methyl		Inorganic		Methyl	
		µg/day	µg/kg/day	µg/day	µg/kg/day	µg/day	µg/kg/day	µg/day	µg/kg/day
Mean	0.55	0.12	0.009	0.49	0.04	2.48	0.04	9.90	0.14
Minimum	0.19	0.04	0.003	0.17	0.01	0.86	0.01	3.42	0.05
Maximum	1.01	0.23	0.02	0.90	0.07	4.55	0.06	18.18	0.26

[1] Reported as total mercury: assumed to be 80% methylmercury and 20% inorganic.
[2] Based on a 13.3 kg child and a daily fish consumption rate of 1.12 g/day.
[3] Based on a 70 kg adult and a daily fish consumption rate of 22.5 g/day.

4.2.2.4.3 Biological monitoring of Great Lake anglers - Cornwall

Biological monitoring of mercury levels in anglers has recently been carried out by Health Canada in the Great Lakes Anglers Pilot Exposure Assessment Study (Kearney and Cole, 1995). Both whole blood and hair were sampled in anglers from Cornwall and Mississauga. Total mercury levels in whole blood from anglers in the Cornwall area (n= 41) were above detection limit (2 ppb) in 73 of 113 of those anglers that consumed their fish and 8 of 28 persons that did not eat fish. Samples ranged from 2 to 17 ppb. All blood levels were below the Health Canada guideline level of 20 ppb. The arithmetic mean mercury level for fish eaters in this study (2.8 ppb) is well below reference values for fish consumers (8.4 ppb). With respect to inorganic mercury, levels were detectable in only 19 of the 232 samples (8%) taken from both areas. Results of statistical modelling suggested blood mercury levels were 35% higher ($p = 0.003$) for sport fish consumers compared with non-consumers.

Hair mercury was also analyzed in 809 samples from both Cornwall and Mississauga. Total mercury was above detection limit (0.5 ppm) in only 36% of all samples and inorganic mercury was detected in only 2% of samples. One participant had a hair level of 8.4 ppm placing him in the "increasing risk" category specified by Health Canada. This individual was a fish eater with a blood mercury level well within the normal range.

4.2.2.5 Ingestion of drinking water - survey data and estimate of mercury exposure

The MOEE monitors drinking water at municipal water supply systems throughout Ontario through the Drinking Water Surveillance Program (DWSP). Samples are taken of raw and treated water at the treatment plant and analyzed for various chemical and microbiological contaminants. Further, water samples at the consumer's tap water are taken to monitor some contaminants which may emanate from the distribution system. In the case of mercury, tap water samples are not evaluated as there is no source of mercury in distribution systems that would introduce mercury to the drinking water. The mercury analysis in raw and treated drinking water does not consider speciation but rather analyses and reports total mercury concentrations. However, it is assumed that mercury present in drinking water is predominantly in its inorganic form.

Cornwall residents are serviced by three water treatment plants (WTP): Charlottenburg, Cornwall and Prescott WTPs. However, the residents of the community in the vicinity of ICI facility are serviced solely by the Cornwall WTP (K. Columbus, 1994, personal communication). Hence, the monitoring data for the Cornwall WTP has been selected to evaluate this population's exposure.

Monitoring results of mercury concentrations in both raw and treated water for the years 1991 through to and including 1993 are shown in Appendix H. Generally, the measured mercury concentrations are all less than the detection limit, 0.02 µg/L, for all three WTPs, for both raw and treated water (MOEE, 1993). The one exception was a February 1993 measurement of raw water at the Charlottenburg WTP of 0.18 µg/L; however, once treated, the mercury level in the drinking water was below the detection limit. The Cornwall WTP was consistently below the detection limit for mercury in drinking water.

From 1991-1993, the concentrations of total mercury measured in treated water at the Cornwall WTP were consistently below the detection limit of 0.02 µg/L. This is well below the current Ontario Drinking Water Objective (ODWO) for inorganic mercury and the Canadian Drinking Water Guideline (CDWG) for total mercury of 0.001 mg/L. Because the concentrations of mercury in water sampled during this survey period are below the limit of detection, the concentration used to assess exposure is half the detection limit, 0.01 µg/L. Average daily intake of drinking water was assumed to be 0.6 L/day for children and 1.5 L/day for adults (Health Protection Branch, 1981). The estimated intake is calculated by solving for the following equation:

$$Intake = C_{water} \times IR$$

where,

C_{water} = concentration of mercury in drinking water (µ/L)
IR = ingestion rate of drinking water

As indicated in Table 4.13, the average estimated intake of mercury in drinking water in the Cornwall area is 0.006 and 0.02 µg/day for children and adults, respectively.

Table 4.13: Estimated intake of Mercury from Drinking Water

Mercury concentration in drinking water (Cornwall WTP) (µg/L)	Estimated intake of mercury from drinking water			
	Child (0.5-4 years)[1]		Adult (>20 years)[2]	
	µg/day	µg/kg/day	µg/day	µg/kg/day
0.01	0.006	0.0005	0.02	0.0003

[1] Based on a 13.3 kg child and a daily water consumption rate of 0.6 L/day.
[2] Based on a 70 kg adult and a daily water consumption rate of 1.5 L/day.

4.2.2.6 Integrated estimates of mercury intake through ingestion

Exposure to mercury is possible through the ingestion of soil, food (including contaminated backyard vegetables and angled fish) and drinking water. Integration of the estimated intakes of mercury from these sources results in a total ingested mercury intake. It should be noted that not included in these ingestion estimates are possible intakes resulting from dental amalgam.

Table 4.14: Integrated Exposure Estimate (Ingestion) - Typical Community Resident - No Backyard Vegetables and No Angled Fish

Media	Estimated intake of mercury through ingestion (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil	0.003	-	0.0001	-
Food - Fish	0.005	0.02	0.004	0.02
Food - Non-Fish	0.24	-	0.11	-
Drinking Water	0.0005	-	0.0003	
Total	**0.2485**	**0.02**	**0.1144**	**0.02**

Table 4.15: Integrated Exposure Estimate (Ingestion) - Typical Community Resident - Backyard Vegetables and No Angled Fish

Media	Estimated intake of mercury through ingestion (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil	0.003	-	0.0001	-
Garden Vegetables	0.04	-	0.01	-
Food - Fish	0.005	0.02	0.004	0.02
Food - Non-Fish	0.24	-	0.11	-
Drinking Water	0.0005	-	0.0003	
Total	**0.2885**	**0.02**	**0.1208**	**0.02**

Table 4.16: Integrated Exposure Estimate (Ingestion) - Typical Community Resident - No Backyard Vegetables, Angled Fish

Media	Estimated intake of mercury through ingestion (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil	0.003	-	0.0001	-
Food - Fish	0.005	0.02	0.004	0.02
Food - Non-Fish	0.24	-	0.11	-
Angled Fish	0.009	0.04	0.04	0.14
Drinking Water	0.0005	-	0.0003	
Total	**0.2575**	**0.06**	**0.1544**	**0.16**

Table 4.17: Integrated Exposure Estimate (Ingestion) - Typical Community Resident - Backyard Vegetables and Angled Fish

Media	Estimated intake of mercury through ingestion (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil	0.003	-	0.0001	-
Garden Vegetables	0.04	-	0.01	-
Food - Fish	0.005	0.02	0.004	0.02
Food - Non-Fish	0.24	-	0.11	-
Angled Fish	0.009	0.04	0.04	0.14
Drinking Water	0.0005	-	0.0003	
Total	**0.2975**	**0.06**	**0.1644**	**0.16**

4.2.3 Dermal exposure

Exposure to mercury in soil/dust through dermal contact may occur through a number of activities including gardening by adults and backyard play by children. Dusts are present on essentially all surfaces (e.g., swings, pavement and buildings) and the amount of contact with the skin will depend on individual behaviour. For most inorganic forms of metals in solution, it is expected that dermal absorption will not occur to a significant extent (see Appendix I for a technical discussion). Inorganics that are soil-bound are expected to be dermally absorbed to a much lesser extent than those in solution. The bioavailability of inorganics on skin (percentage released from the soil matrix during skin contact), together with the limited penetration of the epidermis, suggest that this exposure will be of negligible magnitude.

4.2.3.1 Estimated dermal exposure through direct soil contact

In general, metals are believed to have very little ability to penetrate skin. However, because of some observations suggesting skin absorption of mercury and the known dermatological effects of mercury including contact dermatitis, some crude estimation of dermal exposure is important to carry out. No distinction is made regarding mercury species as little is known

regarding the quantitative aspects of either metallic, inorganic or methylmercury and, as with soil ingestion estimates, all measured mercury is total.

The available data concerning dermal absorption of metallic and inorganic mercury is very sparse. Dermal absorption of metallic mercury vapour in humans occurred at a rate of 0.024 ng per cm^2 of skin per minute per every ng Hg/m^3 of air (Hursh *et al.*, 1989). This was roughly 1% of the uptake rate via inhalation (WHO, 1991). Direct exposure to liquid metallic mercury would be expected to result in higher absorption rates; however, these have not been evaluated in humans. It is also well known that the use of skin-lightening creams containing mercury can cause substantial absorption. Absorption of mercurous salts (which are also likely present in these soils) can occur through skin but no quantitative data exist. It is therefore known that mercury may be absorbed through skin but the rates remain very unclear. Estimation of dermal absorption from mercury adsorbed to soil particles is even more problematic given that absorption of the mercury to organic content of the soil would be expected to significantly limit dermal absorption.

In order to estimate the contribution of the dermal route to exposure, the concentrations of mercury found in the phytotoxicity surveys are used, as well as predicted values utilizing SURFER modelling. The maximum value from 1993 backyard vegetable samples is also used as a worst case maximum.

Estimates of dermal exposure from a contaminant is a complex relationship dependent upon a number of parameters (i.e. size of exposed skin area, contact time, concentration of contaminant in direct contact with skin, soil adhesion , etc). Detailed discussion and derivation of these components have already been done and can be found in the U.S. EPA (1992) document entitled *"Dermal Exposure Assessment: Principles and Applications"*. The calculation of dermally delivered intakes of mercury in residential soil is based on the current U.S. EPA methodology for inorganics. The dermally absorbed dose (DAD; µg/kg/day)) is given by:

$$DAD = \frac{AD_{event} \times EF \times ED \times A}{BW \times AT}$$

where,

EF = event frequency
adults:1 event/day; 40 events/year
children:1 event/day; 150 events per year
ED = event duration
adults:30 years
children:7 years
A = surface area of skin
adults:5000 cm^2 (mean)
children:1800 cm^2 (upper 95th percentile in children 2-4 years old, includes hands, forearms, lower legs and feet)
BW = body weight
adults:70 kg
children:13.3 kg
AT = averaging time for effect
adults:70 years
children:7 years

and where,

AD_{event}= absorbed dose per event (mg/cm^2-event) is given by:

$$AD_{event} = C_{soil} \times AF \times ABS$$

where,
C_{soil} = concentration in soil (mg/kg)
AF = soil adherence factor (mg/cm^2-event)
an upper end value of 1 is selected
ABS = absorption fraction (unitless)

Assumptions selected for these parameters are conservative and in general were selected from the upper end of values recommended by the U.S. EPA dermal assessment methods (U.S. EPA, 1992) and the U.S. EPA Exposure Factors Handbook (U.S. EPA, 1990). The absorption factor (1%) selected was based upon the observations of Hursh *et al.* (1989) in direct vapour absorption studies. This assumption may underestimate the actual dermal intake because liquid mercury will likely have higher absorption rate and may overestimate the dermal exposure because mercury in soil will be largely bound in soil in forms which are much less available for absorption than pure mercury. For these reasons it is thought to be a reasonable value to employ. It also agrees reasonably well with values estimated [calculation not shown] on the basis of prediction utilizing steady-state methods and permeability co-efficients from water. The use of the direct data absorption data is preferred. AD_{event} is calculated to be $5X10^{-8}$ mg/cm^2- event at the

mean soil concentration using these assumptions.

Estimated dermal intakes of mercury in soil are shown in Table 4.18. Dermal exposure ranges from 0.001 µg/kg/day to 0.072 µg/kg/day in adults and from 0.002 µg/kg/day to 0.12 µg/kg/day in children with typical values of 0.015 µg/kg/day in adults and 0.025 µg/kg/day in children. Estimates corresponding to the community areas bounded by the predicted soil contours using SURFER are also provided.

Table 4.18: Estimated dermally absorbed doses of mercury from direct soil contact

Mercury concentration in soil (µg/g)		Estimated Mercury Intake (µg/kg/day)	
		Children (0-4 years)	Adult (>20 years)
Soil Survey Data			
Mean	0.50	0.025	0.015
Minimum	0.04	0.002	0.001
Maximum	2.37	0.12	0.072
Modelled Concentrations (SURFER)			
Contour 1	0.50	0.025	0.015
Contour 2	1.0	0.050	0.030
Contour 3	1.5	0.075	0.038

4.2.4 Exposure via inhalation

4.2.4.1 Measured mercury levels in ambient air in Cornwall

In June 1993, the Ministry's Atmospheric Research and Special Programs Section conducted an ambient air quality survey of total mercury vapour in the vicinity of ICI Forest Products in Cornwall using the mobile Trace Atmospheric Gas Analysis (TAGA) unit (Ng, 1993). The results of this survey are presented in Table 4.19.

Table 4.19: Mercury vapour concentrations in the vicinity of ICI, - June, 1993

Date	Sample #	Site[1]	Concentration (ng/m^3)		Comments
			TWA[2]	Peak[3]	
June 15	S01	A	4.9	5.4	Upwind, heavy rain
	S02	A	4.6	5.3	Upwind
	S03	A	3.7	4.7	Upwind
	S04	B	17	46	Downwind
	S05	B	58	170	Downwind
	S06	B	3.7	6.5	Downwind
	S07	B	2.5	3.5	Downwind
	S08	C	98	98	Downwind, strong odour from Domtar
	S09	C	46	46	Downwind, strong odour from Domtar
	S10	C	140	140	Downwind, strong odour from Domtar
June 16	S11	A	1.9	2.3	Upwind, overcast
	S12	A	2.6	6.1	Upwind
	S13	A	1.8	1.9	Upwind
	S14	A	1.8	2	Upwind
	S15	D	140	220	Downwind
	S16	D	54	150	Downwind
	S17	E	250	490	Downwind
	S18	E	210	440	Downwind
	S19	E	240	350	Downwind
	S20	E	270	390	Downwind
	S21	A	4.4	5.4	Upwind
June 17	S22	E	2.2	2.8	Upwind, sunny
	S23	E	110	240	Downwind
	S24	E	34	92	Downwind
	S25	E	70	140	Downwind, strong odour from Domtar
	S26	E	57	140	Downwind, strong odour from Domtar
	S27	E	47	94	Downwind, overcast
	S28	E	170	330	Downwind
	S29	E	180	260	Downwind
	S30	E	180	260	Downwind
	S31	E	180	290	Downwind
	S32	E	63	150	Downwind
	S33	A	4	4.7	Upwind

Table 4.19: Mercury vapour concentrations in the vicinity of ICI, - June, 1993

Date	Sample #	Site[1]	Concentration (ng/m^3)		Comments
			TWA[2]	Peak[3]	
June 22	S34	A	3.7	5.7	Upwind, heavy rain
	S35	A	2.4	5.5	Upwind
	S36	A	1.8	2	Upwind
	S37	F	430	670	Downwind, consistent WS and WD
	S38	F	340	570	Downwind, strong chlorine odour
	S39	F	250	340	Downwind
	S40	F	310	480	Downwind
	S41	F	380	480	Downwind
	S42	F	390	640	Downwind
	S43	F	370	640	Downwind
	S44	F	110	180	Downwind
	S45	F	310	610	Downwind
	S46	F	130	300	Downwind
	S47	F	670	880	Downwind
	S48	F	740	1000	Downwind
	S49	F	720	990	Downwind
	S50	F	810	1200	Downwind, wood chip odour
	S51	C	4	5.2	Upwind, partly cloudy
	S52	C	3	3.5	Upwind
June 23	S53	C	2.8	3.1	Upwind, sunny and clear
	S54	F	290	420	Downwind
	S55	F	390	590	Downwind
	S56	F	340	440	Downwind
	S57	F	350	490	Downwind
	S58	F	300	390	Downwind
	S59	F	330	540	Downwind
	S60	F	170	250	Downwind
	S61	F	320	680	Downwind
	S62	F	300	480	Downwind
	S63	F	170	460	Downwind
	S64	A	9.3	11	Upwind
June 24	S65	A	3.7	4.4	Upwind, sunny and clear
	S66	E	110	170	Downwind
	S67	E	82	110	Downwind, strong odour from Domtar
	S68	E	48	83	Downwind, strong odour from Domtar
	S69	E	190	420	Downwind, no odour
	S70	E	210	380	Downwind
	S71	E	220	450	Downwind
	S72	E	140	290	Downwind
	S73	E	86	150	Downwind
	S74	E	250	540	Downwind
	S75	E	120	390	Downwind
	S76	E	53	120	Downwind
	S77	A	4.3	5.3	Upwind

[1] See Figure 4 for monitoring locations.
[2] TWA = time weighted average = half-hour average (average of six 5-minute averages).
[3] Peak = highest of the six 5-minute averages.

Monitoring of mercury vapour in this area was also done in 1984 and 1986 and results of these MOEE surveys have been published (De Brou and Chapman, 1985; Chapman, 1987). The equipment used in the 1993 survey is approximately 50 times more sensitive than that used in previous MOEE surveys, allowing for measurement of mercury at lower concentrations (detection limit: 0.13 ng/m^3). Readings were taken every five minutes and half-hour average concentration were calculated using the average of six five minute measurements (Ng, 1993). The analytical method did not allow for speciation, therefore total mercury was measured. It is recognized that greater than 90% of Hg in air is in the elemental form (Mike Corbridge, 1994, personal communication).

The monitoring survey was conducted in the period of June 14-25, 1993. The location of monitoring sites relative to the ICI facility is illustrated in Figure 4.4. All of the monitoring sites were within 1000 meters of the ICI facility. A total of 77 half-hour samples were acquired, consisting of 19 reference (upwind) and 58 downwind samples. As expected, the reference samples generally had lower mercury concentrations than downwind samples. Half-hour average concentrations for upwind sites ranged from 1.8-4.9 ng/m^3, with the exception of one half-hour average of 9.3 ng/m^3 (Site A, sample number S64) taken near a ventilation system of a water purification plant. The half-hour mercury concentrations at sites downwind of ICI had a broader range: 2.5-810 ng/m^3 with a maximum 5 minute measurement of 1200 ng/m^3.

The measurements made by the TAGA group were under daytime conditions, with the associated improved dispersion of gases from the cell room. Emission rate data, as measured by a Federal source monitoring team, in conjunction with the TAGA results was combined into a computer simulation study by the Regional Atmospheric & Terrestrial Effects Unit, MOEE (Ladouceur, 1994). The results of this examination, when extended to the still air conditions of the night, indicated that ambient mercury levels may be higher by a factor of three during the night. Deposition rates will increase accordingly.

The current MOEE 1/2 hour point of impingement guideline is 5000 ng/m^3 (5 µg/m^3) (refer to Regulation 346). All samples had mercury concentrations below the current 1/2 hour point of impingement guideline.

In 1974, the Ministry proposed a 24 hour ambient air quality criterion (AAQC) of 2 µg/m^3 for inorganic Hg, which was 1/25 of the Threshold Limit Value (TLV) proposed by the American Conference of Governmental Industrial Hygienists at that time, and a 24 hour AAQC of 0.5 µg/m^3 for mercury alkyl

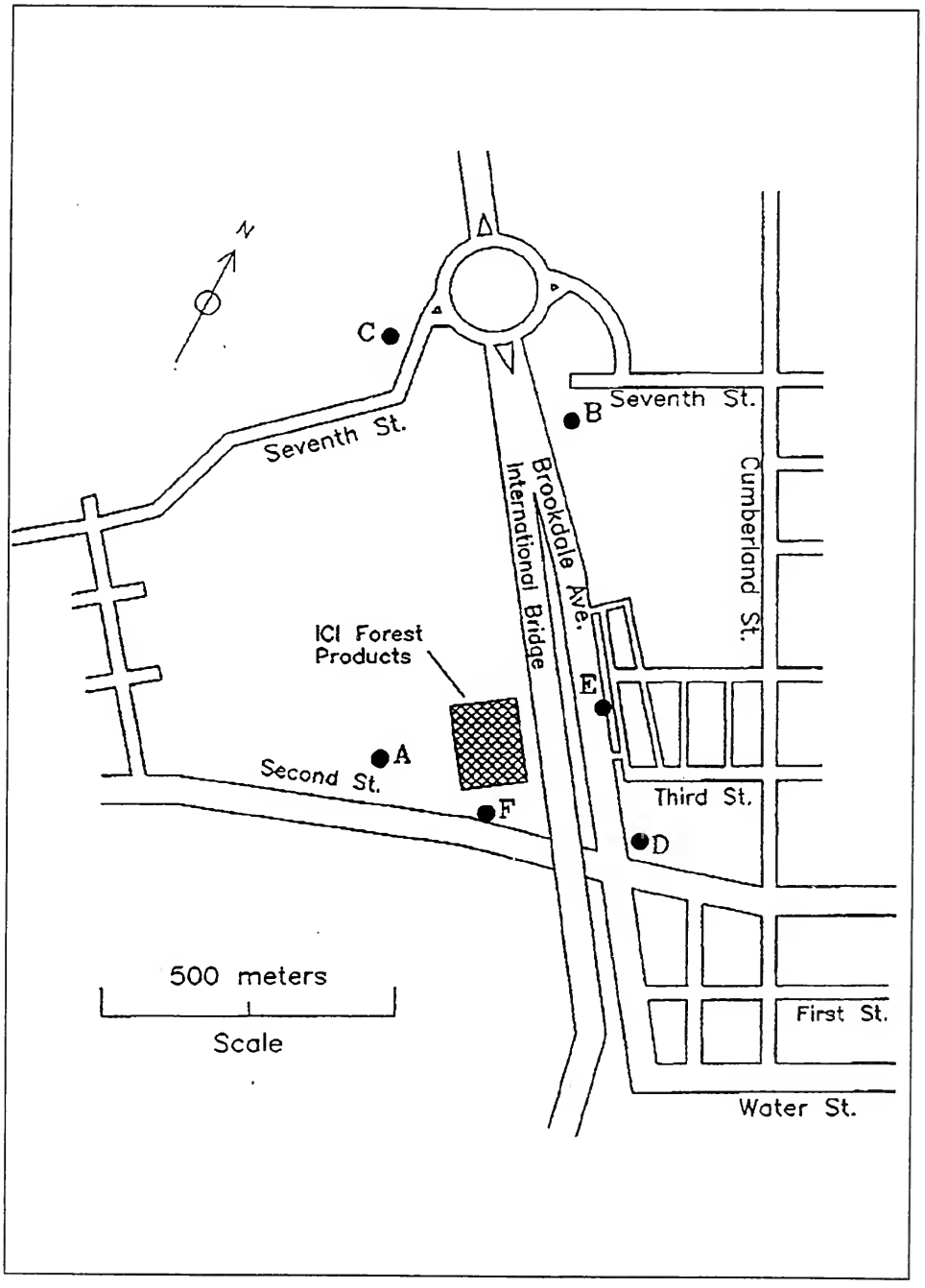

Figure 4.4: Mercury monitoring sites (ambient air) in the vicinity of ICI Forest Products, Inc., Cornwall (June 1993)

compounds, based on the then available TLV-TWA. Both AAQC values are currently under review/update by the Ministry.

Figure 4.5 shows annual and growing season (May - August) cell room mercury emissions to the atmosphere from 1976 to 1994. Mercury emissions show a 74% decrease from 1976 to 1984, the year of lowest total emissions. A 20% increase in emissions occurred from 1984 to 1991. The growing season emissions accounted for, on average, 48% of the total emissions based on emission data from 1976 to 1994. Higher emissions in the summer months may be due to the opening of roof vents to improve ventilation and the increased volatility of mercury in warm weather.

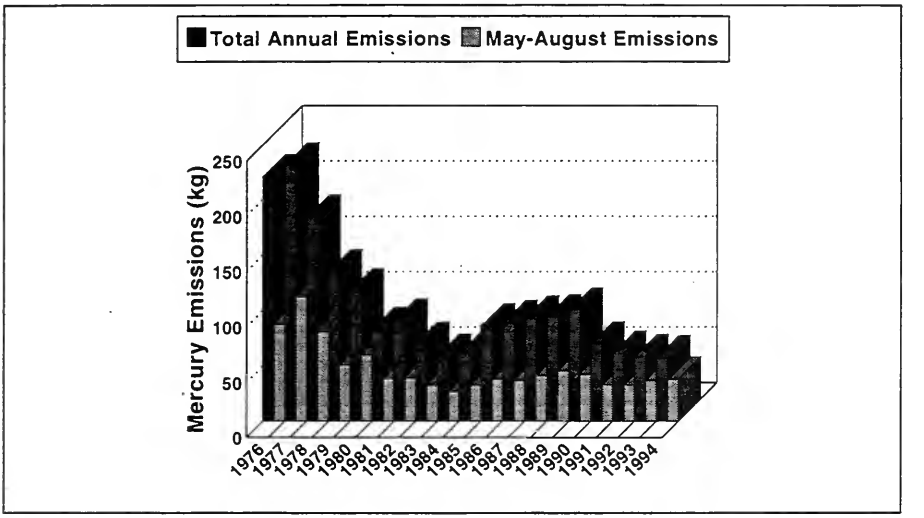

Figure 4.5: ICI Cell Room Mercury Emissions to the Atmosphere (1976-1994). Source: Emerson, 1995 (personal communication)

4.2.4.2 Estimated intake of mercury via inhalation

For the calculation of inhalation intakes of mercury in this community, selected data from the 1993 TAGA survey were utilized. This data is considered the most direct information to use. For the purposes of modelling, only levels reported from sites B, D, and E were selected as these TAGA sites are situated within the residential area itself where higher air concentrations are noted compared to other sites upwind of the ICI facility. From 28 samples taken under various wind conditions, the mean 1/2 hour average is 126 ng/m^3, with a range of 3.7 to 270 ng/m^3.

Estimated intakes are the product of mercury concentration in air and inhalation rates in children and adults. These results are shown in Table 4.20. Typical inhalation exposures are approximately 0.036 µg/kg/day in adults and 0.047 µg/kg/day for a child.

Table 4.20: Estimated Inhalation Intakes of Mercury in the Cornwall Community

Mercury Concentration in Air (TAGA Data) (ng/m^3)		Estimated Mercury Intake (µg/kg/day)	
		Child (0-4 years)	Adult (> 20 years)
Mean	126.6 (n=28; sites B, D, E)	0.047	0.036
Minimum	3.7	0.001	0.001
Maximum	270	0.10	0.076

[1] Based on a 13.3 kg child and an inhalation rate of 5 m^3/day.
[2] Based on a 70 kg adult and an inhalation rate of 20 m^3/day.

4.2.4.3 Airborne mercury exposures in other Ontario cities

In 1992, the MOEE's Mobile Monitoring Group measured ambient levels of gaseous inorganic mercury at 19 sites in Windsor, Ontario (Ng, 1993). Samples were taken over a period of eight days. The site-specific mean ambient mercury concentrations ranged from 1.3 to 52 ng/m^3, while the maxima ranged from 1.6 to 94 ng/m^3. With the exception of the site having the 52 ng/m^3 mean value, all other means were less than 8 ng/m^3. Similarly, with the exception of the site having a maximum of 94 ng/m^3, all other

maxima were less than 16 ng/m^3. Therefore, the mean value of 8 ng/m^3 and a maximum value of 16 ng/m^3 were selected as representative values for the entire urban Windsor area.

4.2.5 Mercury exposure from dental amalgams

Dental amalgam, generally known as silver tooth filling, contains approximately 50% mercury by weight; copper amalgam, used mostly in paediatric dentistry, may contain up to 70% mercury (WHO, 1991). The use of mercury in dental amalgam may result in exposure to dentists, dental assistants and patients receiving amalgam. Given the toxicity of mercury, the safety of mercury-containing dental amalgams has been questioned in numerous articles published in scientific journals and by the general media.

Inorganic mercury vapour emitted from dental amalgam may be absorbed by the patient, mainly through the routes of inhalation or ingestion, resulting in chronic exposure to mercury and possible adverse health effects. The World Health Organization considers dental amalgam to be a significant source of exposure to mercury faced by the general population and estimated the intake of inorganic mercury vapour to be 3.8-21 µg/day (WHO, 1991). Intake of mercury from dental amalgam, including ionic mercury and elemental mercury vapour, has also been estimated at about 23 µg/day for adults and 5.4 µg/day for children aged 0.5 to 4 years (Mitchell, 1992). Some vapour may dissolve in the saliva as inorganic mercury, however, there is no firm evidence for this.

There has been some controversy regarding the health hazard of mercury from dental amalgam. Numerous studies have been done examining the relationship between certain health effects and mercury containing tooth fillings. These studies have been done in dental patients, as well as in dentists and dental workers, who are occupationally exposed to higher levels of mercury than the patients themselves. The WHO concluded, in 1991, that the current scientific evidence regarding health hazards from dental amalgams is as of yet inconclusive (WHO, 1991).

4.3 Estimated mercury exposure following closure of the mercury cell room

A key question which is raised by the announced closure of the mercury cell room at ICI is what level of exposure can be expected associated with the soil contamination. This includes questions as to the safety of consuming vegetables after airborne emissions of mercury form ICI have essentially stopped.

The modelling of this exposure situation is developed utilizing the following assumptions:

Soil: intakes related to soil ingestion and dermal contact will not change for the short and medium-term as soil mercury levels will not vary considerably except perhaps with some expected decrease in surface dust concentration.

Air: the major source of mercury emissions from ICI will have been decommissioned. It is not known whether there will be any significant emissions from other operations in the plant although it is reasonably predictable that air levels of mercury in the community will be very much lower. Previous reviews have indicated average atmospheric concentrations of mercury in air to be about 0.001 to 0.O5 ng/m^3 in Canada and internationally. In the Windsor '91 Risk Assessment, adult and child intakes of gaseous inorganic mercury averaged 0.003 µg/kg/day. The average for the Canadian/Ontario population is 0.018 µg/day (0.0026 µg/kg/day) in adults; 0.036 µg/day (0.0026 µg/kg/day) in young children is assumed. These are in relatively good agreement and therefore a "background" inhalation exposure of 0.003 µg/kg/day is assumed for both adults and children.

- The mercury contamination that remains in soil will be an additional source of mercury in air. For this reason, mercury emission rates and expected concentrations in the community were modelled using the MOEE CAP source dispersion model with the impacted community as the receptor area (see Appendix J). Using very conservative modelling assumptions, and a soil concentration based on the very large 0.5 ppm mercury in soil contour 1, average mercury levels in the air are predicted to be between 2-9 ng/m^3. A level of 9 ng/m^3 is used as the "additional" intake related to the soil source. A worst case estimate of 400 ng/m3 is presented but it is not likely that the emission rate

calculated and climate conditions could reasonably coincide to produce these higher modelled levels.

- the modelling in Appendix J suggests depletion of the soil mercury source within 80 years assuming a 1 meter depth of contamination. Depth soil profiles from the late 1970s (see Appendix K) suggest a significant drop off in contamination at 15 cm depths. It is therefore a more realistic assumption that contamination is to a depth of 15 cm. Therefore the total loss of mercury in this soil is more likely to occur within 12 to 20 years given the predicted emission rates are reasonably accurate.

Food: background mercury exposures in diet (non-site related) are assumed.

Backyard vegetable consumption:

Plants are known to uptake mercury from soil but to a limited extent and this uptake varies with soil concentration. Soil uptake rates of mercury from soil are not well characterized and will depend on a number of soil chemistry and individual crop characteristics. Levels of mercury within backyard vegetables must be estimated based on uptake from soil. Levels in the 1993 backyard vegetable data set will be predominantly due to aerial deposition of mercury. Experiments using lettuce and radish conducted by the MOEE Phytotoxicity section on the ICI property and in the controlled environment facility in Brampton suggest that roughly 91% of total mercury burden is due to airborne emissions and that 8% is due to root uptake from soil. Assuming no change in short to mid-term mercury soil concentrations, estimated vegetables concentrations and resulting intakes using the chronic and acute exposure models are taken at 8% of the values calculated based on 1993 observed dry weight data.

Drinking water: no change due to cell room closure. The same figures are utilized.

4.4 Summary - Discussion of Exposure Calculations

The integrated exposure estimates for inorganic, methyl and total mercury for both adults and children are provided in Tables 4.21-4.32. These integrated intake estimates provide a crude determination of the possible intake of "typical" individuals, as opposed to actual intakes which will vary considerably between individuals depending on behaviour and contact rates with mercury contaminated media. As well, the relative contribution of each route of exposure to total intake is calculated.

A comparison of various exposure scenarios for adults and children are provided in Figures 4.6 and 4.7. These underline the differences between typical Ontario exposures and study area residents (past) and typical study area residents (post closure). Children in the study are predicted to have mercury exposures slightly higher than the typical Ontario child but there is essentially no significant difference in exposure following closure of the mercury cell room and cessation of airborne emissions. Consumption of backyard vegetables increases exposure about 30% above the typical Ontario exposure in children. For adults, the levels of total exposure are roughly half the exposure levels for children because of the difference in body weight. For adults, consumption of sport fish is by far the highest source of mercury intake.

The relative contribution of exposure pathways to total exposure are illustrated in Figures 4.8 and 4.9. The general food basket diet (food, non-fish) as the predominant exposure pathway is very evident. Combining all study area-specific exposures doses not approach this level of intake. Also, it is evident that consumption of locally caught fish results in much larger exposures than any of those associated with the contamination of soil.

Exposure to inorganic and methylmercury occurs for all persons in the general population through its presence in very small amounts in air, soil, dust, food and drinking water. Chronic total mercury intakes for the typical Ontario resident (*i.e.* not living in the study area) are calculated to be 0.27 µg/kg/day for children, with 92% of exposure attributed to inorganic mercury. Average intakes for adults are lower at approximately 0.14 µg/kg/day of which 85% is attributed to inorganic mercury. By far, the greatest contribution to total mercury exposure is food which accounts for roughly 98% of total exposure in typical Ontario adults and young children.

Estimates of typical exposures for residents in the study area (based on past

monitoring) data) suggest an increased previous exposure to inorganic mercury relative to the general Ontario population of roughly 1.3 times for the average child, and 1.4 times for the average adult. The chronic mercury intake for the typical study area resident is estimated to have been 0.34 µg/kg/day for children and 0.19 µg/kg/day for adults. This represents an increase of 0.07 µg/kg/day and 0.05 µg/kg/day above typical Ontario resident exposures, for children and adults, respectively.

By far, the greatest relative contribution to mercury exposure in the study area, as in the general population, is diet. For the typical resident, diet contributes between 70-80% of the total inorganic mercury exposure estimate. Total combined contribution of site-specific media to exposure (ingestion of soil, dermal contact with soil and inhalation of ambient air) is a relatively small fraction of total exposure, accounting for roughly 1/4 of estimated exposure in children and 1/3 of estimated exposure in adults (see Figure 4.8 and 4.9).

Typical inhalation exposures are estimated at approximately 0.1 µg/kg/day and 0.076 µg/kg/day in adults. Measured ambient air concentrations average 127 ng/m^3 and range from 3.7 to 270 ng/m^3. This exposure is about 10 fold higher than might be seen in a typical Ontario city, and accounts for approximately 15-20% of total of total mercury intake.

Dermal contact with contaminated soil may contribute slightly to overall total intake of mercury. According to the exposure model, the estimated intake from dermal contact with soil is about 1/2 of the inhalation intake and about 1/10 of the oral intake associated with general diet. It is unlikely that significant amounts of mercury would be absorbed through the skin because of the binding of the mercury to soil particles.

A number of residents in this area grow and consume backyard vegetables. The consumption of backyard vegetables results in a mean increase to the total mercury intake of 0.04 µg/kg/day and 0.01 µg/kg/day in children and adults, respectively, based on the chronic exposure model. This model assumes that consumption of these vegetables in averaged over the entire year. In actuality, backyard vegetables are consumed in large amounts on a periodic basis, particularly during the summer and fall season of harvest. Acute periodic intakes in inorganic mercury through backyard vegetable consumption would, on average, add 0.15 µg/kg/day in children, and 0.09 µg/kg/day in adults, and under worst case concentrations, as much as 0.52 µg/kg/day in children and 0.33 µg/kg/day in adults. Under these

assumptions, modelling suggests that there is some potential for periodic intakes through eating vegetables that could increase total daily exposures to inorganic mercury by as much as 2 to 3 times compared to those living in the community who do not consume homegrown vegetables. For various reasons the worst case scenarios does not represent the likely exposure for an average child. For the most part very young children do not consume large amounts of backyard vegetables, and when these vegetables are consumed, it is done only periodically.

Consumption of angled fish would increase the total mercury intake for the typical child in the community by 0.049 µg/kg/day and for the adult community resident by 0.18 µg/kg/day, based on calculations using data from the Sport Fish Contaminant Monitoring Program. 80% of this intake is assumed to be in the form of methylmercury and 20% in the form of inorganic mercury. As discussed above, for adults, this is by far a much higher exposure than soil or vegetables would account for. It is also noteworthy that the Health Canada survey of anglers in the Cornwall area did not reveal any consumers of locally caught fish above Health Canada guideline levels and that in most cases, inorganic mercury was not detectable.

Key matters raised by the closure of the mercury cell-room are the potential for mercury exposure following the closure and the potential health risks, if any associated with these exposures. For this reason, a plausible scenario of multimedia exposure was modelled which included off-gassing of mercury form soil to air and uptake of mercury form soil into vegetables. Estimated total mercury intakes for residents under this scenario are not significantly different from those of the general Ontario population at 0.30 µg/kg/day for children and 0.16 µg/kg/day for adults.

A significant decrease in mercury concentrations in ambient air is predicted due to closure of the mercury source. However, some off-gassing of mercury vapour from soil to air is still likely to occur. Ambient air levels following closure of the cell room facility are estimated to be 2-9 ng/m^3 on the basis of soil-to-air emission modelling. These levels are well within the typical range of urban air quality for mercury. There is also no significant exposure associated with backyard vegetable consumption as calculated, based on limited uptake of mercury from soil into plants and a significant reduction of aerial mercury deposition.

Following closure of the cell room facility, it is estimated that mercury in soil

will decrease over time and be modelled to background concentrations through natural degradation processes within 12-20 years. This estimate is based on the modelled source emission rates from soil together with assumptions regarding the depth of contamination (see Appendix J). Correspondingly, exposures should generally decrease over this period. Field studies of soil depth profiles would be required to verify these assumptions.

Table 4.21: Integrated Exposure Estimates of Mercury Intake - Typical Ontario/Canadian

Media	Estimated intake of mercury (µg/kg/day)[1]			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil	0.0003	0	0.00001	0
Food				
Fish	0.005	0.02	0.004	0.02
Non- Fish	0.24	0	0.11	0
Drinking Water	0.002	0.0008	0.0009	0.0003
Air	0.0032	0.0008	0.0021	0.0006
Total	**0.25**	**0.02**	**0.12**	**0.02**
	0.27		**0.14**	

[1] The values in Table 4.21 do not include exposure from dental amalgams. The current database is not adequate to accurately estimate such exposure. Based on limited data and assumptions, intake of elemental mercury vapour from dental amalgams could be 14 µg/d and intake of ionic mercury could be 9 µg/day for adults. For children, intake of elemental mercury from dental amalgams could be 3.5 µg/day and intake of ionic mercury could be 1.9 µg/day. Such exposures would represent a major contribution to total mercury exposure, but may be overestimates for children as they are likely to have few amalgam restorations.

Modified from Mitchell (1992)

Table 4.22: Relative Contribution of Exposure Pathways to Total Mercury Intake (by species) - Typical Ontario/Canadian

Media	Relative Contribution (%)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil	0.12	-	0.01	-
Food				
Fish	2.00	92.59	3.42	95.69
Non- Fish	95.81	-	94.01	-
Drinking Water	0.80	3.70	0.77	1.44
Air	1.28	3.70	1.79	2.87
Total (numbers in brackets indicate % of total mercury)	**100 (92.06)**	**100 (7.94)**	**100 (84.85)**	**100 (15.15)**
	100		**100**	

Table 4.23: Integrated Exposure Estimates of Mercury Intake -Typical Community Resident

Media	Estimated intake of mercury through ingestion (μg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.003	-	0.0001	-
Dermal	0.025	-	0.015	
Food				
Background Food - Fish	0.005	0.02	0.004	0.02
Background Food - Non-Fish	0.24	-	0.11	-
Drinking Water	0.0005	-	0.0003	-
Air	0.047		0.036	
Total	**0.32**	**0.02**	**0.17**	**0.02**
	0.34		**0.19**	

Table 4.24: Relative Contribution of Exposure Pathways to Total Mercury Exposure (by species) - Typical Community Resident

Media	Relative Contribution (%)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.94	-	0.06	-
Dermal	7.80	-	9.07	
Food				
Background Food - Fish	1.56	100	2.42	100
Background Food - Non-Fish	74.88	-	66.51	-
Drinking Water	0.16	-	0.18	-
Air	14.66		21.77	
Total	**100 (94.13)**	**100 (5.87)**	**100 (89.21)**	**100 (10.79)**
(numbers in brackets indicate % of total mercury)	**100**		**100**	

Table 4.25: Integrated Exposure Estimates of Mercury Intake -Typical Community Resident - Backyard Vegetables

Media	Estimated intake of mercury through ingestion (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.003	-	0.0001	-
Dermal	0.025	-	0.015	
Food				
Background Food - Fish	0.005	0.02	0.004	0.02
Background Food - Non-Fish	0.24	-	0.11	-
Garden Vegetables	0.04	-	0.01	-
Drinking Water	0.0005	-	0.0003	-
Air	0.047	-	0.036	-
Total	**0.36**	**0.02**	**0.18**	**0.02**
	0.38		**0.20**	

Table 4.26: Relative Contribution of Exposure Pathways to Total Mercury Exposure (by species) - Typical Community Resident - Backyard Vegetables

Media	Relative Contribution (%)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.83	-	0.06	-
Dermal	6.93	-	8.55	
Food				
Background Food - Fish	1.39	100	2.28	100
Background Food - Non-Fish	66.57	-	62.71	-
Garden Vegetables	11.10	-	5.70	-
Drinking Water	0.14	-	0.17	-
Air	13.04	-	20.52	-
Total	**100 (94.74)**	**100 (5.26)**	**100 (89.76)**	**100 (10.24)**
(numbers in brackets indicate % of total mercury)	**100**		**100**	

Table 4.27: Integrated Exposure Estimates of Mercury Intake -Typical Community Resident - Angled Fish

Media	Estimated intake of mercury through ingestion (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.003	-	0.0001	-
Dermal	0.025	-	0.015	
Food				
Background Food - Fish	0.005	0.02	0.004	0.02
Background Food - Non-Fish	0.24	-	0.11	-
Angled Fish	0.009	0.04	0.04	0.14
Drinking Water	0.0005	-	0.0003	-
Air	0.047	-	0.036	-
Total	**0.33**	**0.06**	**0.21**	**0.16**
	0.39		**0.37**	

Table 4.28: Relative Contribution of Exposure Pathways to Total Mercury Exposure (by species) - Typical Community Resident - Angled Fish

Media	Relative Contribution (%)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.91	-	0.05	-
Dermal	7.59	-	7.30	
Food				
Background Food - Fish	1.52	33.33	1.95	12.50
Background Food - Non-Fish	72.84	-	53.55	-
Angled Fish	2.73	66.67	19.47	87.50
Drinking Water	0.15	-	0.15	-
Air	14.26	-	17.53	-
Total	**100 (84.60)**	**100 (15.40)**	**100 (56.21)**	**100 (43.79)**
(numbers in brackets indicate % of total mercury)	**100**		**100**	

Table 4.29: Integrated Exposure Estimates of Mercury Intake - Typical Community Resident - Backyard Vegetables and Angled Fish

Media	Estimated intake of mercury through ingestion (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.003	-	0.0001	-
Dermal	0.025	-	0.015	
Food				
Background Food - Fish	0.005	0.02	0.004	0.02
Background Food - Non-Fish	0.24	-	0.11	-
Garden Vegetables	0.04	-	0.01	-
Angled Fish	0.009	0.04	0.04	0.14
Drinking Water	0.0005	-	0.0003	-
Air	0.047		0.036	
Total	**0.37**	**0.06**	**0.22**	**0.16**
	0.43		**0.38**	

Table 4.30: Relative Contribution of Exposure Pathways to Total Mercury Exposure (by species) - Typical Community Resident - Backyard Vegetables and Angled Fish

Media	Relative Contribution (%)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.81	-	0.05	-
Dermal	6.77	-	6.96	
Food				
Background Food - Fish	1.35	33.33	1.86	12.50
Background Food - Non-Fish	64.95	-	51.07	-
Garden Vegetables	10.83	-	4.64	-
Angled Fish	2.44	66.67	18.57	87.50
Drinking Water	0.14	-	0.14	-
Air	12.72	-	16.71	-
Total	**100 (94.74)**	**100 (5.26)**	**100 (57.38)**	**100 (42.62)**
(numbers in brackets indicate % of total mercury)	**100**		**100**	

Table 4.31: Integrated Exposure Estimates of Mercury Intake - Typical Community Resident Consuming Backyard Vegetables - Cell Room Closure

Media	Estimated mercury intake (µg/kg/day)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	0.003	-	0.0001	-
Dermal	0.025	-	0.015	-
Food				
Background Food- Fish	0.005	0.02	0.004	0.02
Background Food - Non-Fish	0.24	-	0.11	-
Backyard Vegetables	0.0032	-	0.008	-
Drinking Water	0.0005	-	0.0003	-
Air				
Background	0.003	-	0.003	-
Soil Vapour	0.002	-	0.001	-
Total	**0.28**	**0.02**	**0.14**	**0.02**
	0.30		**0.16**	

Table 4.32: Relative Contribution of Exposure Pathways to Total Mercury Exposure (by species) - Typical Community Resident Consuming Backyard Vegetables - Cell Room Closure

Media	Relative Contribution (%)			
	Child (0.5-4 years)		Adult (>20 years)	
	Inorganic	Methyl	Inorganic	Methyl
Soil				
Ingestion	1.06	-	0.07	-
Dermal	8.87	-	10.61	-
Food				
Background Food- Fish	1.77	100	2.83	100
Background Food - Non-Fish	85.20	-	77.79	-
Backyard Vegetables	1.14	-	5.66	-
Drinking Water	0.18	-	0.21	-
Air				
Background	1.06	-	2.12	-
Soil Vapour	0.71	-	0.71	-
Total	**100 (93.37)**	**100 (6.63)**	**100 (87.61)**	**100 (12.39)**
(numbers in brackets indicate % of total mercury)	**100**		**100**	

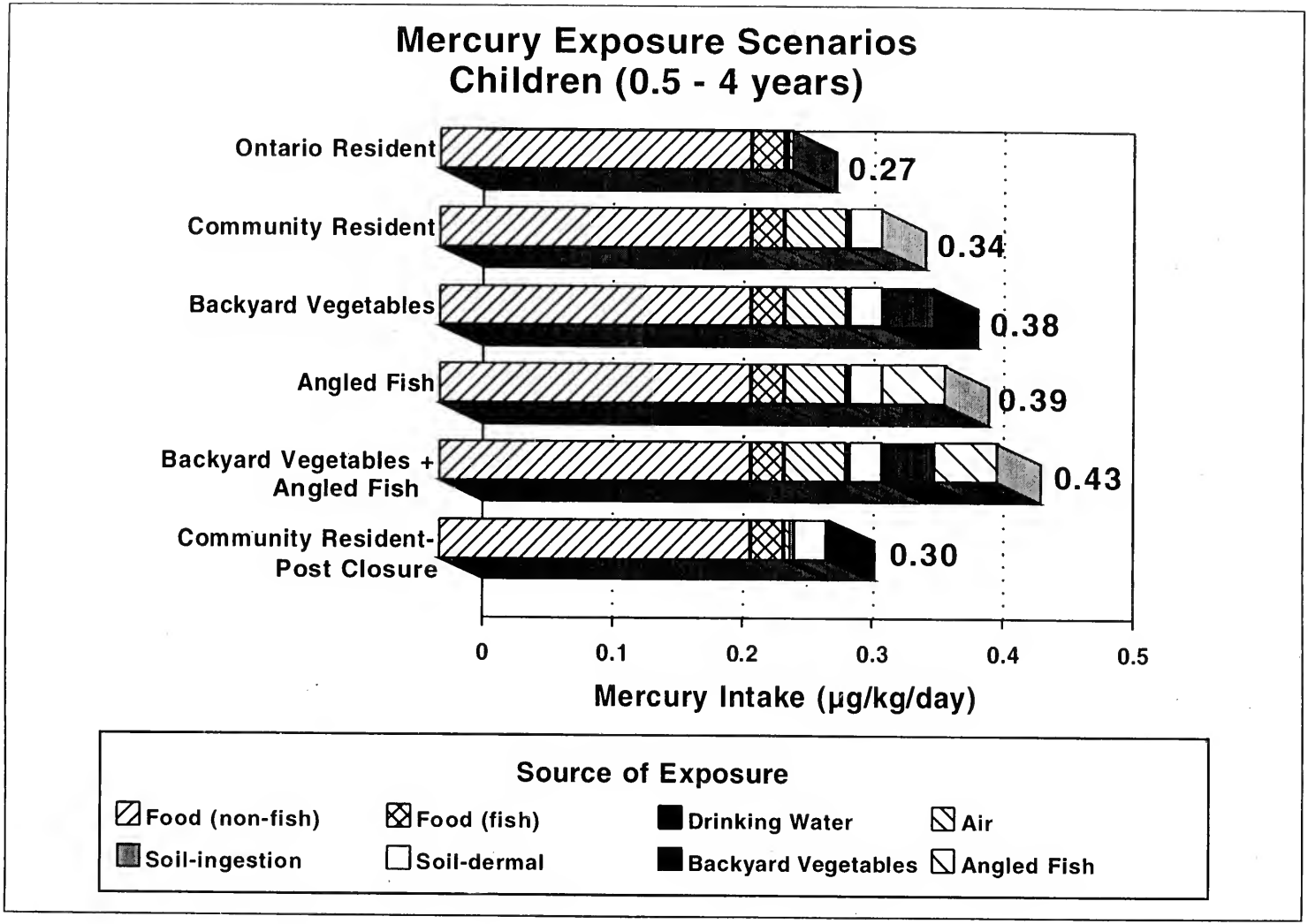

Figure 4.6: Mercury exposure scenarios for children (0.5-4 years).

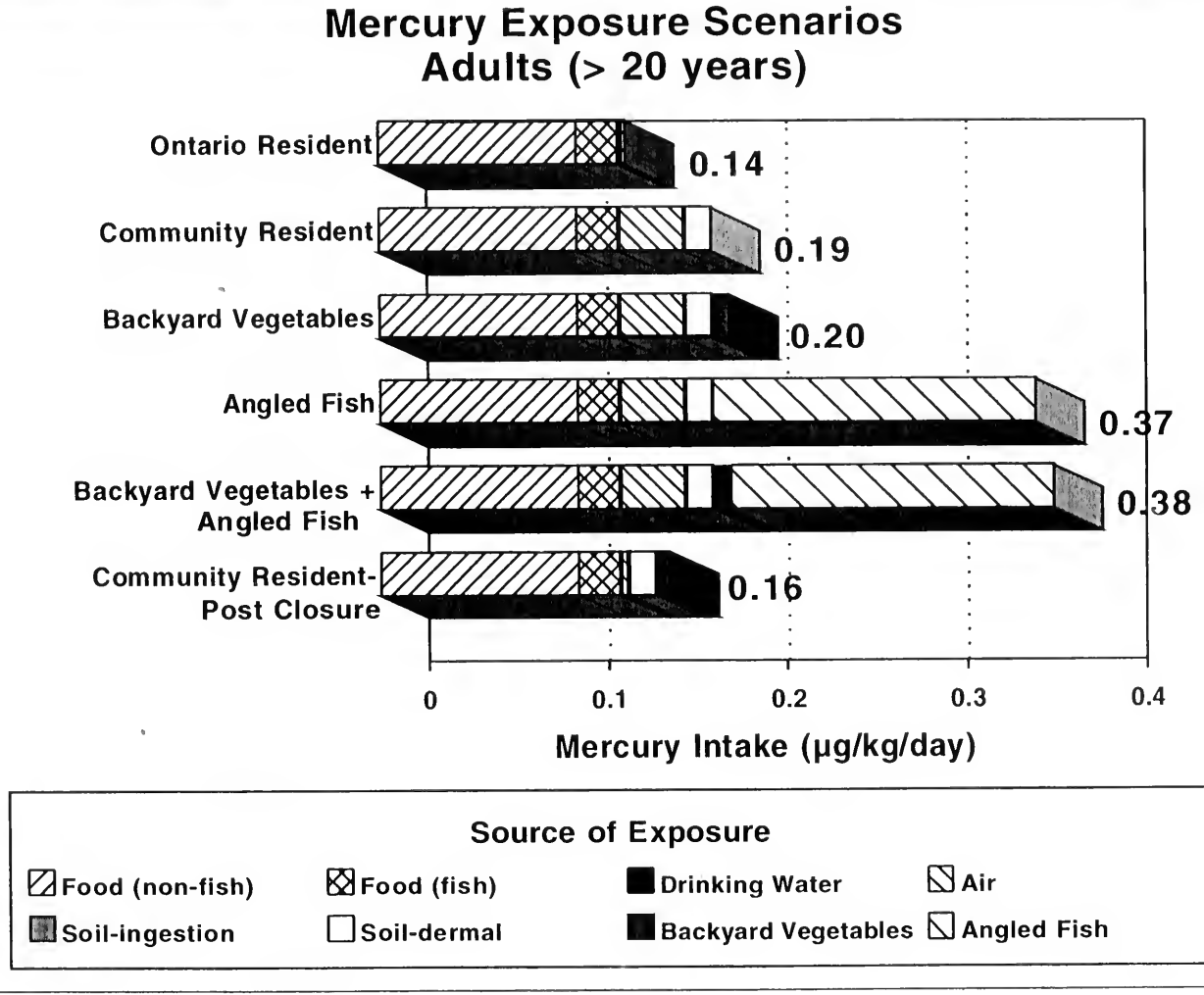

Figure 4.7: Mercury exposure scenarios for adults (>20 years).

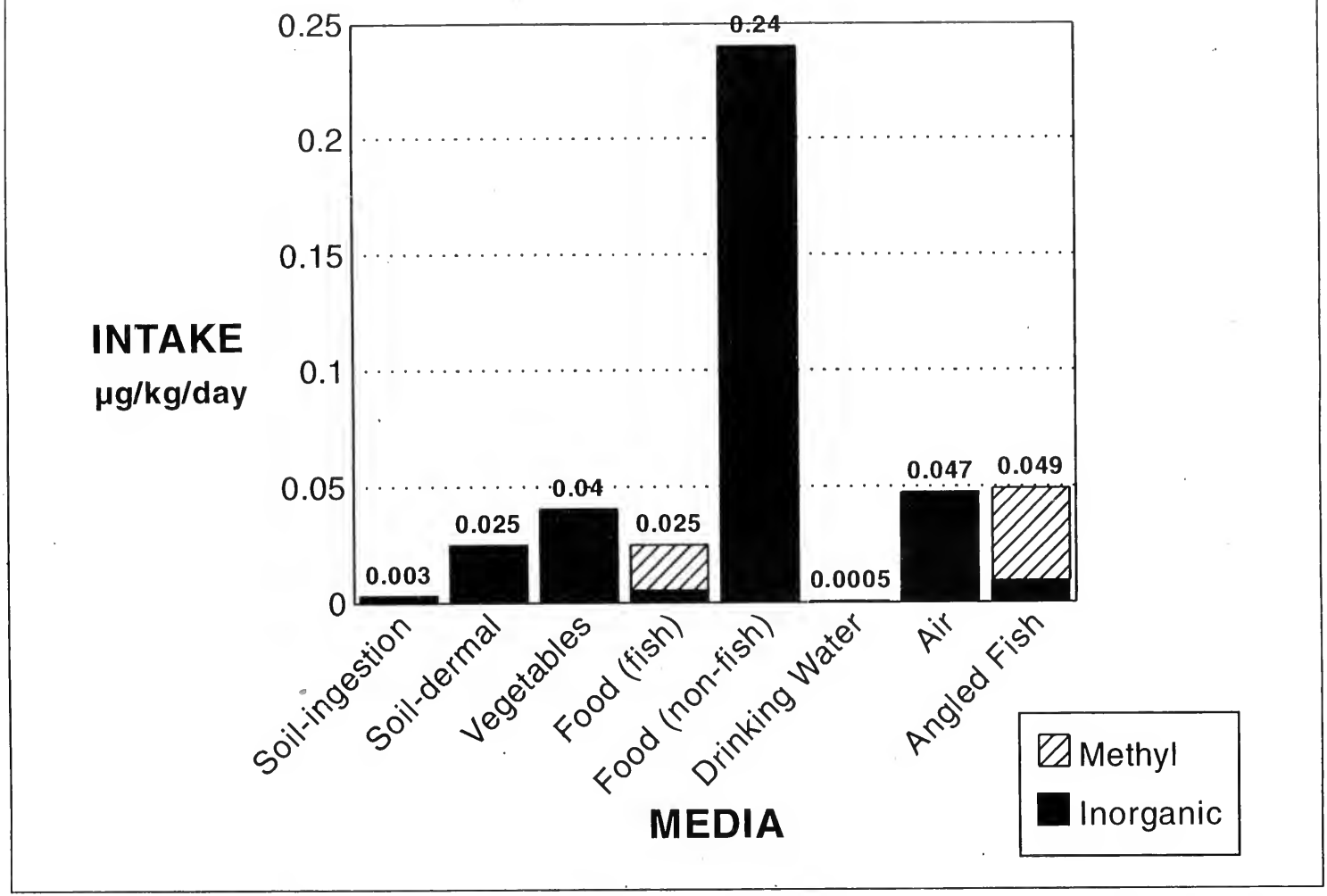

Figure 4.8: Estimates of mercury intake by media for children (0.5-4 years).

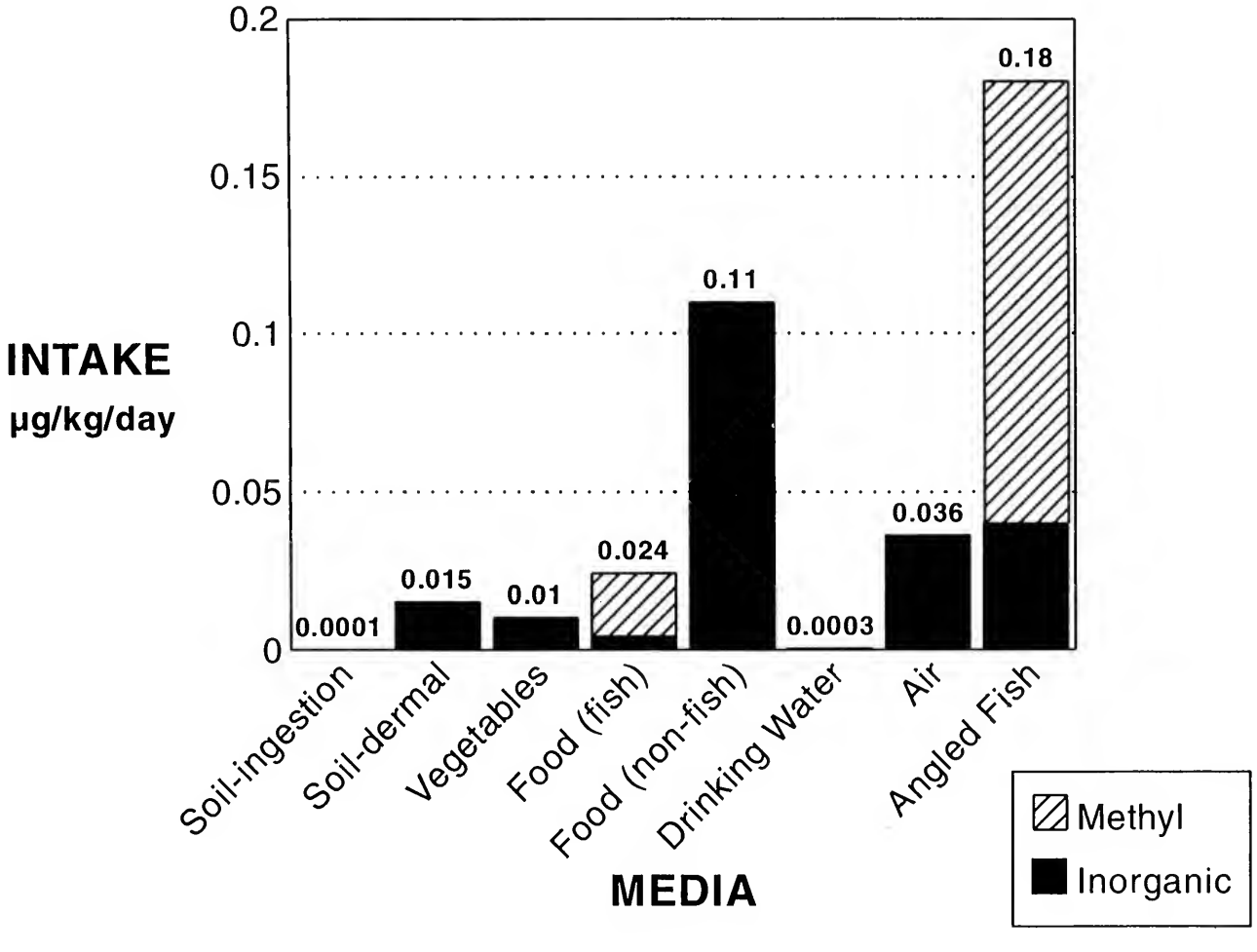

Figure 4.9: Estimates of mercury intake by media for adults (>20 years).

5.0 RISK CHARACTERIZATION

In the risk characterization stage, estimated exposures under various scenarios are compared with health criteria and dose-response information to provide a quantitative picture of potential risk. Risk characterization for mercury is complicated by a variety of factors including the role of mercury speciation in the production of specific types of adverse effects, the need to examine mercury exposure by route as well as total mercury exposure, and the lack, in this case, of mercury data reported by species.

In carrying out this analysis, several types of health criteria were used based on either total exposure or a specific route of exposure. Level and type of criteria also vary with the form of mercury considered. These are summarized in Table 5.1 below.

Table 5.1: Mercury Health Criteria for Characterizing Risk

	INORGANIC	METHYL	TOTAL
ORAL	0.3 µg/kg/day (EPA Reference Dose) 7 µg/kg/day (ATSDR-Minimal Risk Level, Acute) 2 µg/kg/day (ATSDR - Minimal Risk Level, 6 month)	0.3 µg/kg/day (EPA Reference Dose) 0.48 µg/kg/day (WHO) 0.12 µg/kg/day (ATSDR, Minimal Risk Level, 6 months)	300 µg/week (No more than 200 µg /week methyl) (WHO -total exposure)
INHALATION	340 ng/m^3 (US EPA HEAST- Reference Concentration) 19 µg/kg/day (WHO - equivalent to LOAEL) 14 ng/m^3 ATSDR-Minimal risk level Chronic)	Not currently available	340 ng/m^3 (Acceptable Exposure Level - CALIFORNIA)
DERMAL	NONE	NONE	NONE

Comparisons of ingestion, inhalation and total mercury exposures against current health criteria are shown in Figures 5.1, 5.2 and 5.3. Examining intake of total mercury, typical exposures for children and adults within this community are and have been well below the permissible tolerable weekly intake set by the WHO (see Figure 5.2).

The total child and adult exposures via ingestion including background dietary exposure are well below (less than 10%) of the ATSDR minimum risk level of 2 µg/kg/day as well as below the U.S. EPA oral reference dose of 0.3 µg/kg/day. Under a worst case assumption of maximum measured soil concentration, ingestion exposure is only marginally increased and is still below the most conservative health criteria for oral exposure.

Estimated dermal exposures are small and not anticipated to result in dermatological effects, generally associated with much higher exposures such as would be encountered in occupational exposures to mercury. Thus, exposures associated with soil in this area do not constitute an undue health risk.

The chronic model of backyard vegetable exposure estimated a mean increase of 0.04 µg/kg/day mercury intake in children and 0.01 µg/kg/day in adults, as compared to other residents who do not eat these vegetables. These intakes, on a chronic basis, would not sufficiently add to exposure and exposures would still be within published health criteria for chronic long-term exposure. Total exposure via ingestion for a typical child (not eating backyard vegetables) is close to, but does not exceed, the U.S. EPA oral reference dose for inorganic mercury. Therefore, additional exposures above the typical exposures must be closely examined.

In actuality, vegetables are consumed in large amounts on a periodic basis particularly during the summer and fall season of harvest. Acute periodic intakes in organic mercury through backyard vegetable consumption would, on average, add 0.15 µg/kg/day in children, and 0.09 µg/kg/day in adults, and under worse case concentrations, as much as 0.52 µg/kg/day in children and 0.33 µg/kg/day in adults. Under these assumptions, modelling suggests that there is some potential for periodic intakes through eating vegetables that could increase total daily exposures to inorganic mercury as much as 2-3 X greater than those living in the study area but who do not consume vegetables in this community.

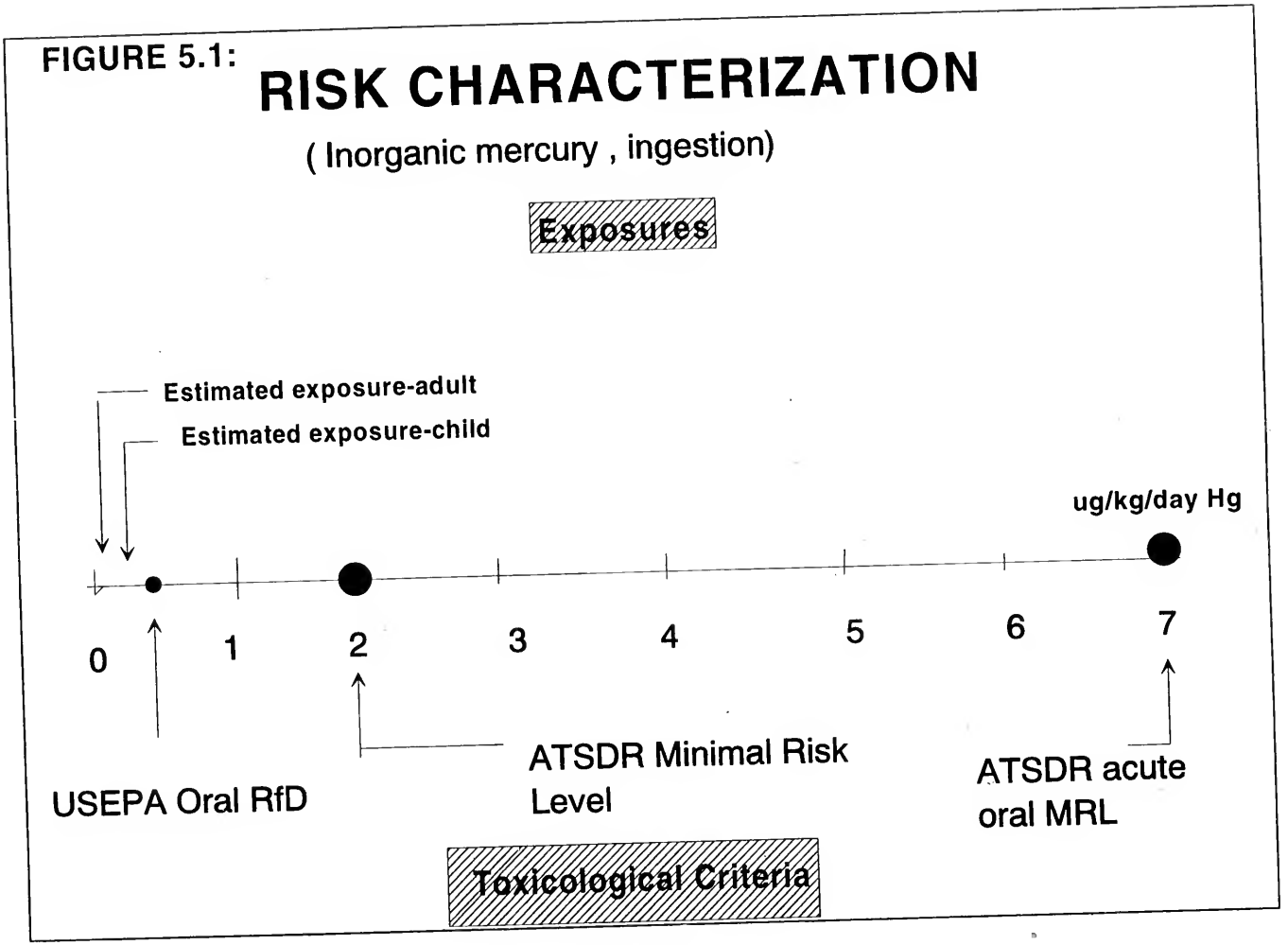

Figure 5.1: Risk Characterization (Inorganic mercury, ingestion).

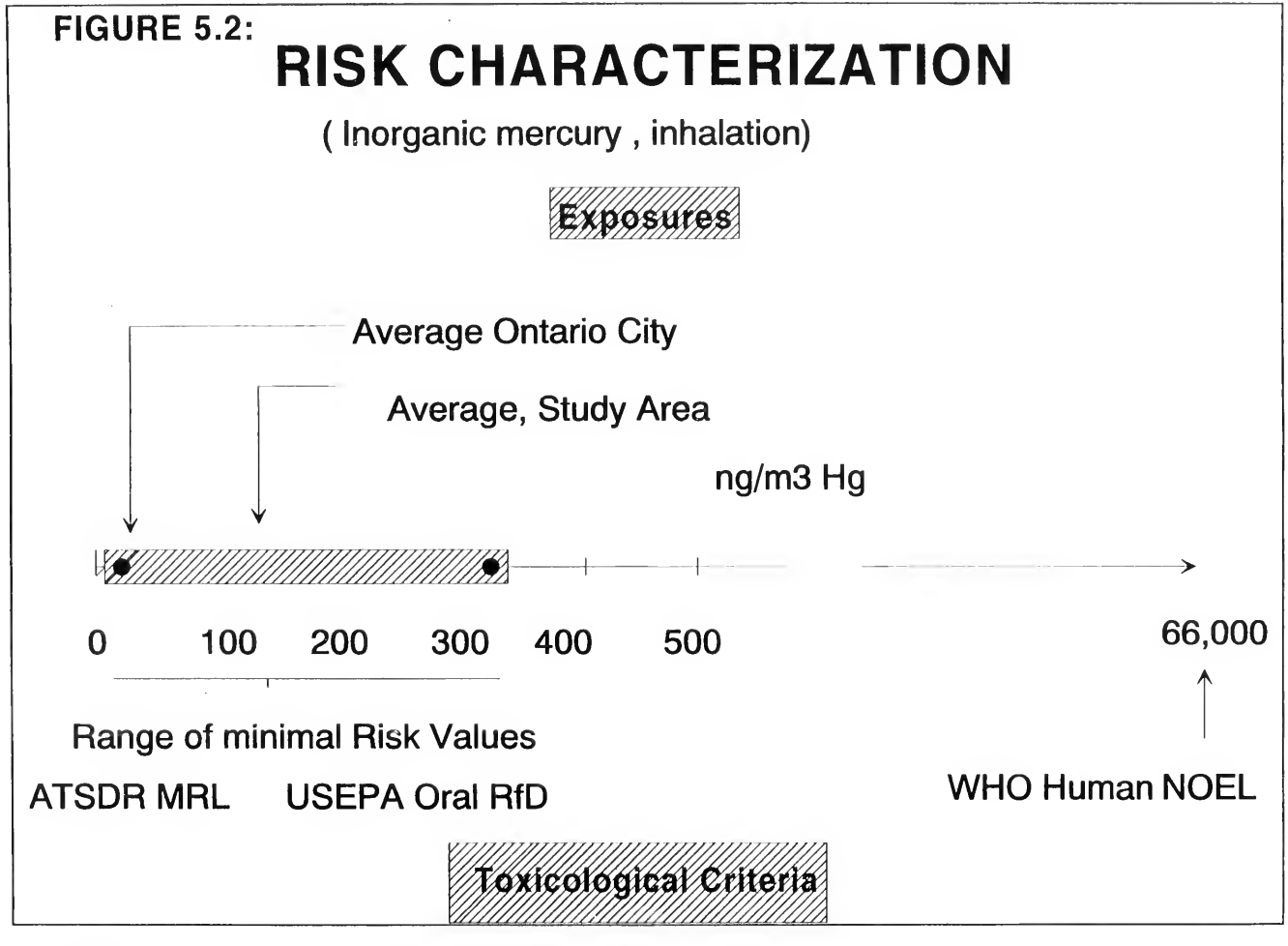

Figure 5.2: Risk Characterization (Inorganic Mercury, inhalation).

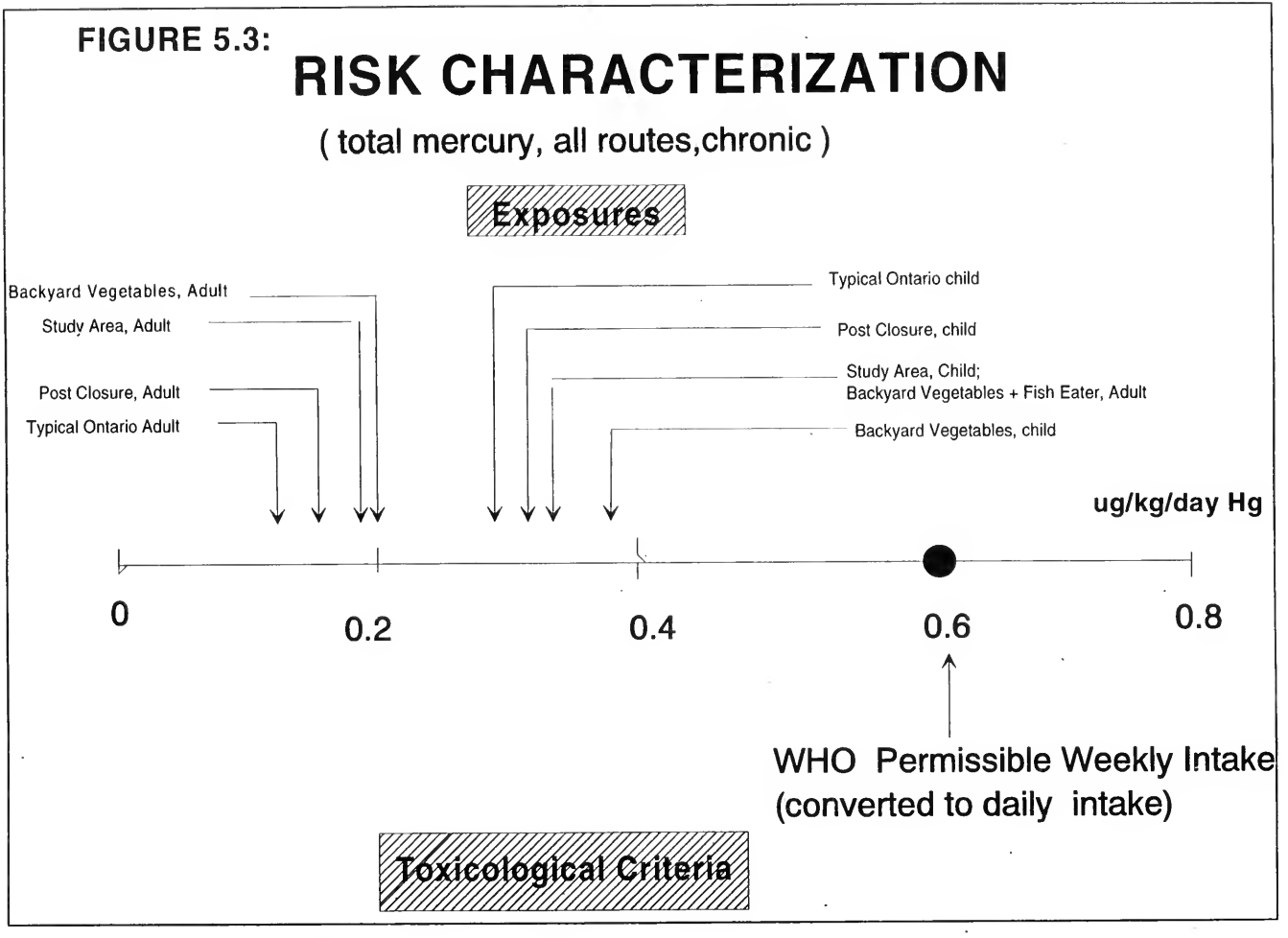

Figure 5.3: Risk Characterization (total mercury, all routes, chronic).

On average, the increased chronic intake of mercury through consumption of vegetables would increase chronic daily intake in children to marginally below the U.S. EPA reference dose (which contains a safety factor of 1000 in its derivation). Adult exposures would also remain below this health criteria. A maximally exposed child, assuming the very highest measured mercury concentration in vegetation, may have some potential for exposures slightly greater than the chronic oral ingestion reference doses, although this would not generally be expected to be the case.

Modelled periodic short-term exposures to mercury through eating vegetables are well below the ATSDR Minimal Risk Level for acute oral exposure and intermediate exposure. Therefore, it is unlikely that these exposures constitute an imminent health hazard. These additional intakes provided by backyard vegetable consumption at the maximum reported mercury concentration could result in exposures which marginally exceed the WHO permissible weekly intake for total mercury. However because the form of mercury is largely inorganic the WHO guideline is not entirely applicable. Also, the worst case exposure is expected to be unlikely and would require a child to eat 100 grams of vegetables at the highest measured concentrations every day of the week. These findings suggest that backyard vegetable consumption during the years of cell room emissions may have resulted in increased mercury intakes for some individuals, most notably young children. These exposures do not present an imminent health hazard. However, it cannot be categorically determined that these short term exposures would not be without some amount of additional risk. This risk is likely to be very small, given that on a chronic basis backyard vegetable consumption accounts for only 11% total exposure in children and approximately 1/6 of the mercury intake compared to background diet intake. Also, there is a very large margin of safety (uncertainty factor of 1,000) contained within the U.S. EPA reference dose derivation. On balance, it would appear that although the additional exposure associated with eating vegetables is unlikely to be associated with adverse effect in children, prudence would suggest that this exposure be avoided under circumstances of continuing emissions from the ICI facility.

It should also be noted that the types of vegetables will to a certain degree dictate levels of exposure. In general, consumption of root vegetables would be associated with much lower exposures than consumption of leafy vegetables. Vegetables sampled were beet top, lettuce and tomato whereas younger children often prefer carrots, peas, beans, and corn. Less leafy vegetables (*i.e.* those with less surface on which to capture aerial deposition

of mercury) would likely have less mercury content.

Average ambient air concentrations and maximum concentrations are below the inhalation chronic acceptable exposure level set by the California Air Toxics Hot Spots Program. All values are also significantly below the reference concentration suggested in the U.S. EPA HEAST Guidelines. Average air concentrations in the study area are higher than typical Ontario urban levels but within the range of published air values associated with minimal risk (see Figure 5.3). When compared to the human inhalation exposure equivalent of the Lowest Observed Adverse Effect Level (LOAEL) for renal effects (WHO, 1991), these calculated intakes are roughly 500 X lower than this particular value. Adverse health impacts are not anticipated at these air levels for the following reasons: *1.)* comparison to existing "minimal risk" criteria and the 1991 WHO human effects level for renal effects; *2.)* the large numerical adjustments (safety factors) in derivation of the minimal risk levels (at least 100 fold); *3.)* air levels are within the 1993 California air guidelines; *4.)* air levels are within the existing U.S. EPA inhalation reference dose (although this is currently under review); *4.)* total exposures are within acceptable criteria for total exposure by all routes; and *5.)* inhalation exposure is less than 1/5 of total exposure and significantly less than normal dietary exposure. Because total exposures are within existing health criteria it is unlikely that these air levels would significantly increase mercury levels in the body or be anticipated to result in adverse effect. The significance of these exposures is complicated by the evolving development of various health criteria by various agencies, using different assumptions and slightly different data sets.

Dermal contact with contaminated soils may contribute slightly to overall total intakes of mercury. According to the model, the estimated intakes are about 1/2 of the inhalation intakes and about 1/10 of the oral intake associated with general diet. It is unlikely that significant amounts of mercury would be absorbed through the skin because of the binding of the mercury to soil particles which occurs. The levels of mercury in soil are unlikely to result in any localized dermal effects as there are no reported cases anywhere of this sort of effect resulting from contact with mercury contaminated soil, and the levels are much lower than those found in situations where a contact dermatitis or sensitization would be expected to occur.

No exposure to methylmercury is associated with the soil or vegetable contamination in the study area. The adult subpopulation with the greatest exposure to mercury includes those individuals consuming angled sport fish.

In this case, methylmercury constitutes a significant fraction of total mercury exposure. For these individuals, the amount of inorganic mercury added by exposure to site-specific mercury source (*i.e.* soil, air, backyard vegetables, drinking water) is a relatively small fraction of the non-site related mercury exposures. In other words, the potential exposure and risk is greatest for fish eaters, and these exposures are much greater than any other exposures that might occur due to contamination of residential soil and vegetables. Consumption of angled fish would increase the total mercury intake for the typical adult community resident by 0.14 μg/kg/day, based on calculations using data from the Sport Fish Contaminant Monitoring Program. 80% of this intake is assumed to be in the form of methylmercury and 20% in the form of inorganic mercury. A recent Health Canada study on fish consumers which included the Cornwall area indicated no individuals with mercury levels above Health Canada guidelines. By corollary, it may be suggested that it is unlikely that any of the exposure scenarios examined would lead to unacceptable blood or hair mercury levels in study area residents.

No untoward exposure or health risk is anticipated following closure of the mercury cell room at ICI as the source of airborne emissions of mercury because:

- total estimated mercury intakes for residents under this scenario are essentially the same as those of the general Ontario population;

- there is also no significant exposure associated with backyard vegetable consumption as calculated based on the uptake of mercury from soil into plants;

- ambient air levels following closure of the cell-room facility are estimated at 2-9 ng/m^3 on the basis of soil-to-air emission modelling, levels which are well within the typical range of urban air quality for mercury.

- following closure of the cell room facility, it is predicted that mercury in soil will decrease over time and be reduced to background levels through natural processes within 12-20 years. This estimate is based upon the modelled source emission rates from soil together with assumptions regarding the depth of contamination. Correspondingly, exposures should generally decrease over this period. Field studies of soil profiles of depth would be required to verify these assumptions.

In summation, exposure to inorganic or total mercury for typical community residents integrated through all pathways are below existing health criteria and is not anticipated to result in adverse effects. This conclusion is also supported by the observation that there have been no identified health effects identified with environmental emissions from chloralkali facilities for mercury in other locations and that all soil levels are below the currently recommended (health-based) decommissioning guideline for mercury in soil of 10 ppm. Persons living in this study area will have had mercury exposures somewhat greater than those of the general population, but would receive the majority of this exposure from background diet and not from site related contamination. Estimated exposure for recreational anglers is greater than any site-related exposures. Adults who engage in recreational angling as well as consume backyard vegetables, will have the highest overall exposures although these are unlikely to result in levels which would exceed Health Canada guidelines for acceptable human tissue levels. For children, consumption of contaminated backyard vegetables may potentially lead to notable increases in exposure particularly at the highest measured concentrations, and therefore, this exposure should be avoided for young children. For future consumption, exposure scenarios suggest that both children and adults can safely consume normal amounts of these background vegetables provided that they are thoroughly washed during preparation to remove dirt particles.

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APPENDIX A

DERIVATION AND SIGNIFICANCE OF THE MOEE PHYTOTOXICOLOGY "UPPER LIMITS OF NORMAL" CONTAMINANT GUIDELINES

Appendix A: Derivation and Significance of the MOEE Phytotoxicology "Upper Limits of Normal" Contaminant Guidelines.

The MOEE Upper Limits of Normal (ULN) contaminant guidelines represent the expected maximum concentration in surface soil, foliage (trees and shrubs), grass, moss bags, and snow from areas in Ontario not exposed to the influence of a pollution source. Urban ULN guidelines were based on samples collected from urban centres, whereas rural ULN guidelines were developed from non-urbanized areas. Samples were collected by Phytotoxicology staff using standard sampling procedures (reference: Ontario Ministry of the Environment and Energy 1992, Phytotoxicology Field Investigation Manual). Chemical analyses were conducted by the MOEE Laboratory Services Branch.

The ULN is the arithmetic mean plus three standard deviations of the suitable background data for each chemical element and parameter. This represents 99% of the sample population. This means that for every 100 samples that have not been exposed to a pollution source, 99 will fall within the ULN.

The ULNs do not represent maximum desirable or allowable limits. Rather, they are an indication that concentrations that exceed the ULN may be the result of contamination from a pollution source. Concentrations that exceed the ULNs are not necessarily toxic to plants, animals, or people. Concentrations that are below the ULNs are not known to be toxic.

ULNs are not available for all elements. This is because some elements have a very large range in the natural environment and the ULN, calculated as the mean plus three standard deviations, would be unrealistically high. Also, for some elements, insufficient background data is available to confidently calculate ULNs. The MOEE Phytotoxicology ULNs are constantly being reviewed as the background environmental data base is expanded. This will result in more ULNs being established and may amend existing ULNs.

APPENDIX B

DOCUMENTATION OF U.S. EPA ORAL REFERENCE DOSE FOR INORGANIC (MERCURIC) MERCURY - DRAFT

Appendix B: Documentation of U.S. EPA Oral Reference Dose for Inorganic (Mercuric) Mercury - Draft

1.(a) REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfDo)

Substance Name -- Mercury (Mercuric); Mercuric Chloride
CASRN -- 7487-94-7
Preparation Date -- 11/01/88

1.(a)(1) ORAL RfD SUMMARY

CRITICAL EFFECT	EXPERIMENTAL DOSES*	UF	MF	RfD
Autoimmune effects Rat Subchronic Feeding and s.c. studies U.S. EPA 1987	NOAEL : none LOAEL:0.266 mg/kg/day LOAEL:0.317 mg/kg/day LOAEL:0.633 mg/kg/ day	1000	1	3E-4 mg/kg/day

* This RfD is based on the back calculations from a Drinking Water Equivalent Level (DWEL) recommended to and subsequently adopted by the U.S. EPA Office of Research and Development and Office of Drinking Water in October, 1987, of 0.010 mg/L: (RfD = 0.010 mg/L x 2 L/day/70 kg bw = 0.0003 mg/kg bw/day). The LOAEL exposure levels, utilized in the three studies selected as the basis of the recommended DWEL, are from Druet *et al.* (1978), Bernaudin *et al.* (1981) and Andres (1984), respectively.

1.(a)(2) PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. EPA 1987. Peer Review Workshop on Mercury Issues. October 26-27, 1987, Summary Report. Environmental Criteria and Assessment Office, Cincinnati, OH.

On October 26-27, 1987 a panel of mercury experts met at a Peer Review Workshop on Mercury Issues in Cincinnati, Ohio and reviewed outstanding issues concerning the health effects and risk assessment of inorganic mercury (U.S. EPA, 1987). Five consensus conclusions and recommendations were agreed to as a result of this workshop and are presented in Table 1. Three studies using the Brown Norway rat as the test strain were chosen, from a larger selection of studies, as the basis for the panel's recommendation of 0.010 mg/L as the DWEL for inorganic mercury. The three studies are presented below for the sake of completeness. However, it must be kept in mind that the recommended DWEL of 0.010 mg/L and back calculated oral RfD of 0.0003 mg/kg/day were arrived at from an intensive review and workshop discussions of the entire inorganic mercury database, not just from one study.

TABLE 1
Consensus Conclusions and Recommendations Agreed on during the Peer-Review Workshop on Mercury Issues*

- The most sensitive adverse effect for mercury risk assessment is formation of mercuric-induced autoimmune glomerulonephritis. The production and deposition of IgG antibodies to the glomerular basement membrane can be considered the first step in the formation of this mercuric-mercury-induced autoimmune glomerulonephritis.

- The Brown Norway rat should be used for mercury risk assessment, the Brown Norway rat is a good test species for the study of Hg2+-induced autoimmune glomerulonephritis. The Brown Norway rat is not unique in this regard (this effect has also been observed in rabbits).

- The Brown Norway rat is a good surrogate for the study of mercury-induced kidney damage in sensitive humans. For this reason, the uncertainty factor used to calculate criteria and health advisories (based on risk assessments using the Brown Norway rat) should be reduced by 10-fold.

- Hg2+ absorption values of 7% from the oral route and 100% from the subcutaneous route should be used to calculate criteria and health advisories.

- A Drinking Water Equivalent Level (DWEL) of 0.010 mg/L was recommended based on the weight of evidence from the studies using Brown Norway rats and limited human tissue data.

* **Source: U.S. EPA 1987**

In the Druet *et al.* (1978) study, the duration of exposure was 8-12 weeks: s.c. injection was used instead of oral exposure. In this study the development of kidney disease was evaluated. In the first phase the rats developed anti-GBM antibodies. During the second phase, which is seen after 2-3 months, the patterns of fixation of antisera changed from linear to granular as the disease progressed. The immune response was accompanied by proteinuria and in some cases by a nephrotic syndrome.

Both male and female Brown Norway rats 7-9 weeks of age were divided into groups of 6-20 animals each. The numbers of each sex were not stated. The animals were injected s.c. with mercuric chloride 3 times weekly for 8 weeks, with doses of 0, 100, 250, 500, 1000 and 2000 µg/kg bw. An additional group was injected with a 50 µg/kg dose for 12 weeks. Antibody formation was measured by the use of kidney cryostat sections stained with a fluorescent sheep anti-rat IgG antiserum. Urinary protein was assessed by the bluret method (Druet *et al.* 1978).

Tubular lesions were seen at the higher dose levels. Proteinuria was seen at doses of 100 µg//kg and above, but not at 50 µg/kg. Proteinuria was considered a highly deleterious effect in that affected animals developed hypoalbuminemia and many died. Fixation of IgG antiserum was detected in all groups except controls (Druet *et al.*, 1978).

Bernaudin *et al.*, (1981) reported that mercurials administered by inhalation or ingestion to Brown Norway rats developed a systemic autoimmune disease. The mercuric chloride ingestion portion of the study involved the forcible feeding of either 0 or 3000 µg/kg bw/week of mercuric chloride to male and female Brown Norway rats for up to 60 days. No abnormalities were reported using standard histological techniques in either experimental or control rats. Immunofluorescence histology revealed that 80% (4/5) of the mercuric exposed rats were observed with a linear IgG deposition in the glomeruli after 15 days of exposure. After 60 days of mercuric chloride exposure, 100% (5/5) of the rats were seen with a mixed linear and granular pattern of IgG deposition in the glomeruli and granular IgG deposition in the arteries. Weak proteinuria was observed in 60% (3/5) of the rats fed mercuric chloride for 60 days. The control rats were observed to have no deposition of IgG in the glomeruli or arteries as well as normal urine protein concentrations.

Andres (1984) administered mercuric chloride (3 mg/kg bw in 1 mL of water) by gavage to five Brown Norway rats and two Lewis rats twice a week for 60 days. A sixth Brown Norway rat was given only 1 mL of water by gavage twice a week for 60 days. All rats had free access to tap water and pellet food. After 2-3 weeks of exposure the Brown Norway mercuric chloride-treated rats started to lose weight and hair. Two of the mercuric chloride treated Brown Norway rats died 30-40 days after beginning the study. No rats were observed to develop detectable proteinuria during the 60- day study. The kidneys appeared normal in all animals when evaluated using standard deposits of IgG present in the renal glomeruli of only the mercuric treated Brown Norway rats. The Brown Norway treated rats

were also observed with mercury-induced morphological lesions of the ileum and colon with abnormal deposited of IgA in the basement membranes of the intestinal glands and of IgG in the basement membranes of the lomina propria. All Observations in the Lewis rats and the control Brown Norway rat appeared normal.

1.(a)(3) UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 1000. An uncertainty factor of 1000 was applied to the animal studies using Brown Norway rats as recommended in U.S. EPA (1987).

MF = 1.

1.(a)(4) ADDITIONAL COMMENTS (ORAL RfD)

Kazantzis *et al.* (1962) performed renal biopsies in 2 (out of 4) workers with nephrotic syndrome who had been occupationally exposed to mercuric oxide, mercuric acetate and probably mercury vapours. They felt that the nephrotic syndrome seen in 3 of the 4 workers may have been an idiosyncratic reaction since many other workers in a factory survey had similarly high levels of urine mercury without developing proteinuria. This conclusion was strengthened by work in Brown Norway rats indicating a genetic (strain) susceptibility and that similar mercury-induced immune system responses have been seen in affected humans and the susceptible Brown Norway rats (U.S. EPA 1987.

The only chronic ingestion study designed to evaluate the toxicity of mercury salts was reported by Fitzhugh *et al.* (1950). In this study, rats of both sexes (20-24/group) were given 0.5, 2.5, 10 40, or 160 ppm mercury as mercuric acetate in their food for up to 2 years. Assuming food consumption was equal to 5% bw/day, the daily intake would have been 0.025, 0.125, 0.50, 2.0 and 8.0 mg/kg bw for the five groups respectively. At this highest dose level a slight depression of body weight was detected in male rats only. The statistical significance of this body weight depression was not stated. Kidney weights were significantly ($p<0.05$) increased at the 2.0 and 8.0 mg/kg bw dose levels. Pathological changes originating in the proximal convoluted tubules of the kidneys were also noted with more severe effects in females than males. The primary weaknesses of this study were the lack of reporting which adverse effects were observed with which dosing groups and that the most sensitive strain, the Brown Norway rat, was not used for evaluating the mercury-induced adverse health effects.

Gale and Ferm (1971) studied the teratogenic effects of mercuric acetate on Syrian golden hamsters. Single doses of 2, 3 or 4 mg/kg were injected i.v. on day 8 of gestation. Growth retardation, increased resorption rates and edema of the fetuses were found at all three dose levels, while an increase in the number of abnormalities was detected at the two higher doses. In a more recent study, Gale (1981) compared the embryotoxic effects of a single s.c. dose of 15 mg/kg

mercuric acetate on the eighth day of gestation in five inbred strains and one non-inbred strain of Syrian hamsters. While strain differences were apparent, a variety of abnormalities were reported in all the strains. Gale (1974) also compared the relative effectiveness of different exposure routes in Syrian hamsters. The following sequence of decreasing efficacy was noted for mercuric acetate; 1.p. > 1.v. > s.c. > oral. The lowest doses used, 2 mg/kg for 1.p and 4 mg/kg for the other three routes, were all effective in causing increased resorption and percent abnormalities.

In male mice administered a single 1.p. dose of 1 mg/kg mercuric chloride, fertility decreased between days 28 and 49 post-treatment with no obvious histological effects noted in the sperm (Lee and Dixon 1975). The period of deceased fertility indicated that spermatogonia and premeiotic spermatocytes were affected. The effects were less severe than following a similar dose of methyl mercury. A single 1.p. dose of 2 mg/kg mercury chloride in female mice resulted in a significant decrease in the total number of implants, number of living embryos and a significant increase in the percentage of dead implants (Suter, 1975). These effects suggest that mercury may be a weak inducer of dominant lethal mutations.

1.(a)(5) CONFIDENCE IN THE ORAL RfD

Study : N/A
Database : High
RfD : High

No one study was found adequate for deriving an oral RfD, but based on the weight of evidence from the studies using Brown Norway rats and the entirety of the mercuric mercury database, an oral RfD of high confidence results.

1.(a)(6) EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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U.S. EPA 1987. Peer Review Workshop on Mercury Issues. October 26-27, 1987, Summary Report. Environmental Criteria and Assessment Office, Cincinnati, OH.

Agency RfD Work Group Review: 08/05/85, 02/05/86, 08/19/86, 11/16/88
Verification Date : 11/16/88

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APPENDIX C

SUMMARY OF CURRENT REGULATORY STANDARDS, GUIDELINES AND CRITERIA FOR MERCURY IN SOIL, WATER, AIR AND FOOD

Appendix C: Summary of Current Regulatory Standards, Guidelines and Criteria for Mercury in Soil, Water, Air and Food

Regulations, guidelines, standards, and objectives regarding mercury in various media have been established by various regulatory agencies. These are summarized in Tables 1 through 5, and organized by media : water, food, air/occupational exposure, soil/sludge/compost food, and release into the environment. Where available, the basis for the limit has been included. Information for these tables was obtained from the following sources:

Agency for Toxic Substances and Disease Registry (ATSDR), 1993. Toxicological Profile for Mercury. U.S. Public Health Service.

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WHO Regional Office for Europe. (1987). Air Quality Guidelines for Europe, European Series No. 23. Copenhagen: WHO Regional Publications.

TABLE 1 MERCURY REGULATIONS - Water

AGENCY	MEDIA	LIMIT	DESCRIPTION	BASIS	LEGISLATION/REPORT
World Health Organization	drinking water	0.001 mg/L	· Guidelline Value for drinking water - intended for use by countries as a basis for the development of standards	· human health	Guidelines for drinking-water quality: Recommendations 1, 1984 Geneva, Switzerland: WHO
Health Canada	drinking water	0.001 mg/L	· Maximum Acceptable Concentration (legally enforceable under appropriate provincial agency)	· human health	Guidelines for Canadian Drinking Water Quality, 1978 (updated 1988)
Ontario Ministry of Environment & Energy	drinking water	0.001 mg/L	· Maximum Acceptable Concentration based on Canadian Drinking Water Guidelines	· human health	Ontario Water Resources Act; Ontario Drinking Water Objectives, 1993
Ontario Ministry of Environment & Energy	ambient water	0.2 µg/L	· Provincial Water Quality Objective - for surface water and ground water where it discharges to the surface	· protective of groundwater, ecosystem health, recreational uses	Water Management: Policies, Guidelines, Provincial Water Quality Objectives of the MOEE, 1994
U.S. EPA	drinking water	0.002 mg/L (inorganic Hg)	· Maximum Contaminant Level (equal to Maximum Contaminant Level Goal)	· based on DWEL of 0.01 mg/L and assumed drinking water contribution of 20% · takes into account occurrence, relative source contribution factors, treatment technology, monitoring capability, costs and human health effects	Safe Drinking Water Act, 1986
		10 µg/L	· Drinking Water Equivalent Level	· human health	
		2 µg/L (inorganic Hg)	· Lifetime Health Advisory	· human health	
U.S. EPA	ambient water	144 ng/L	· Ambient Water Quality Criteria based on consumption of contaminated aquatic organisms and water	· human health	Clean Water Act; Ambient Water Quality Criteria, 1986
		146 ng/L	· Ambient Water Quality Criteria based on consumption of contaminated aquatic organisms only		

AGENCY	MEDIA	LIMIT	DESCRIPTION	BASIS	LEGISLATION/REPORT
Florida	drinking water	0.002 mg/L	· Maximum Contaminant Level	· takes into account occurrence, relative source contribution factors, treatment technology, monitoring capability, costs and human health effects	Public Drinking Water Systems, 1984
New York	drinking water	0.002 mg/L	· Maximum Contaminant Level	· takes into account occurrence, relative source contribution factors, treatment technology, monitoring capability, costs and human health effects	Drinking Water Regulations
New York	ambient water	2 µg/L	· Ambient Water Quality Standard- applied to surface waters which may be used as a source of drinking water (basis of effluent limitations for use in state "Pollutant Discharge Elimination System" permits)	· human health	Water Quality Regulations
New York	ground water	0.002 mg/L	· Ground Water Quality Standard- for waters used as a source of potable water, agricultural water or replenishment of surface waters	· human health	Water Quality Regulations
New Jersey	ground water	0.002 mg/L	· ground water used as a source of potable water, agricultural water, industrial water supply, replenishment of surface waters or other reasonable beneficial uses	· aesthetics, human health	
European Economic Community	drinking water	1 µg/L	· Maximum Admissible Concentration (intended for use by members of the EEC as a basis for the development of their own standards)	· human health	EEC Drinking Water Directive, 1980

TABLE 2 MERCURY REGULATIONS - Food

AGENCY	MEDIA	LIMIT	DESCRIPTION	BASIS
Codex Alimentarious Commission of the Food and Agriculture Organization of the UN/WHO	food	5.0 µg/kg (total Hg) 3.3 µg/kg (methyl Hg)	· Provisional Tolerable Weekly Intake (PTWI)	-
Health Canada	food (fish -excluding swordfish and shark)	0.5 ppm (0.2 ppm for frequent eaters of fish)	· administrative guideline	· human health
Food and Drug Administration	food (bottled water)	0.002 mg/L	· permissible level in bottled water	-

TABLE 3 MERCURY REGULATIONS - Air/Occupational Exposure

AGENCY	MEDIA	LIMIT	DESCRIPTION	BASIS	LEGISLATION/REPORT
World Health Organization	indoor air	1 $\mu g/m^3$	· annual average · Guideline Value for indoor pollution	· human health	WHO Air Quality Guidelines for Europe, 1987
Ministry of Labour	air	0.05 mg/m^3 (total Hg) 0.01 mg/m^3 (Hg-alkyl compounds) 0.15 mg/m^3 (Hg- except alkyl) 0.03 mg/m^3 (Hg-alkyl compounds) 0.15 mg/m^3 (total Hg)	· Time Weighted Average Exposure Value · Short Term Exposure Value · Ceiling Exposure Value	· human health	
U.S. Occupational Safety and Health Administration	air	0.01 mg/m^3 (Hg-alkyl compounds) 0.05 mg/m^3 (mercury vapour) 0.1 mg/m^3 (Hg-aryl & inorganic) 0.03 mg/m^3 (Hg-alkyl compounds)	· Permissible Exposure Limit Time Weighted Average (skin designation) · Ceiling (skin designation) · Short Term Exposure Limit (skin designation)	· human health (neurotoxicity)	
American Conference of Governmental Industrial Hygienists	air	0.01 mg/m^3 (Hg-alkyl compounds) 0.05 mg/m^3 (all forms except alkyl vapour) 0.10 mg/m^3 (aryl and inorganic compounds) 0.03 mg/m^3 (Hg-alkyl compounds)	·Threshold Limit Value Time Weighted Average · Short Term Exposure Limit (skin designation)	· human health (neurotoxicity)	guideline
U.S. National Institute for Occupational Safety and Health	air	0.05 mg/m^3 (Hg vapour) 0.01 mg/m^3 (Hg-alkyl compounds) 28 mg/m^3 (Hg-vapour) 10 mg/m^3 (Hg-alkyl compounds)	· Threshold Limit Value Time Weighted Average · Immediately Dangerous to Life of Health	· human health (neurotoxicity)	guideline
U.S. EPA	air	0.95 $\mu g/m^3$ (Hg-vapour) 0.48 $\mu g/m^3$ (Hg-inorganic) 0.05 $\mu g/m^3$ (Hg-alkyl)	· Ambient Air Level Guideline 8-hour Time Weighted Average based on Threshold Limit Value	· human health	

TABLE 4 MERCURY REGULATIONS - Soil/Sludge/Compost

AGENCY	MEDIA	LIMIT	DESCRIPTION	BASIS	LEGISLATION/REPORT
Ministry of Environment & Energy	sewage sludge	0.1 µg/g 0.5 µg/g 0.8 kg/ha	· mean metal content in uncontaminated soils · maximum permissible metal content in sludged soils · maximum permissible metal addition to uncontaminated soil	-	Guidelines for Sewage Sludge Utilization on Agricultural Lands, 1992
Ministry of Environment & Energy	soil	10 ppm	· site clean up criteria for Ontario surface soil (Agricultural Land Use, Residential Parkland Land Use, Industrial Commercial land Use)	-	Proposed Guidelines for the Clean-Up of Contaminated Sites in Ontario, July 1994
Ministry of Environment & Energy	aquatic sediment	0.2 µg/g (Lowest Effect Level) 2 µg/g (Severe Effect Level)	· Provincial Sediment Quality Guidelines	· protection of aquatic organisms	Guidelines for the Protection and Management of Aquatic Sediment Quality in Ontario, 1993
Ministry of Environment & Energy	compost	0.15 ppm 0.5 ppm 0.5 ppm	· maximum concentration for regular compost · maximum concentration for controlled compost · maximum concentration in soil resulting from use of controlled compost	- - -	Interim Composting Guidelines
European Economic Community	soil, sewage sludge	1.0-1.5 mg/kg 16-25 mg/kg 0.1 kg/ha	· Limit Value for concentration of Hg in soil · Limit value for concentration of Hg in sludge for use in agriculture · limit value for amount of Hg which may be added annually to agricultural land (based on 10 year average)0	-	IRPTC

TABLE 5 MERCURY REGULATIONS - Release into the environment

AGENCY	MEDIA	LIMIT	DESCRIPTION	BASIS	LEGISLATION/REPORT
Environment Canada	liquid effluent	2.5 g Hg/day/tonne of chlorine produced, multiplied by the plant's reference production rate	· limit of discharge of Hg in liquid effluent from chlor-alkali plants	· protection of fish	Fisheries Act, Chlor-Alkali Mercury Liquid Effluent Regulations
Environment Canada	air emissions	5 g Hg/day/1000 kg rated capacity 0.1 g Hg/day/1000 kg rated capacity 1.68 kg Hg/day	· limit of release of Hg in ventilation gases exhausted from cell rooms of chlor-alkali plants · limit of release of Hg in the hydrogen gas stream originating from the denuders of chlor-alkali plants or in ventilation gases exhausted from end boxed and tanks or in gases exhausted from retorts of chlor-alkali plants · limit of total amount of Hg released into the ambient air by a plants from all above sources	-	Canadian Environmental Protection Act, Chlor-Alkali Mercury Release Regulations
Ontario Ministry of Environment & Energy	air emissions	5 $\mu g/m^3$ (Hg) 1.5 $\mu g/m^3$ (Hg - alkyl compounds) 2 $\mu g/m^3$ (Hg) 0.5 $\mu g/m^3$ (Hg- alkyl compounds)	· 1/2 hour limit point of impingement standard · 24 hour Ambient Air Quality Criterion	· human health	Environmental Protection Act, Regulation 296)
U.S. EPA Office of Air Quality Planning and Standards	air emissions	2300 g Hg maximum per 24 hours 3200 g Hg maximum per 24 hours	· emissions from mercury ore processing facilities and mercury cell chlor-alkali plants · emissions from sludge incineration plants, sludge drying plants, or a combination of these that process wastewater treatment plant sludges	-	ATSDR
U.S. EPA	release into the environment	1 lb	· reportable quantity for release in the environment	· aquatic toxicity (aquatic 96h MTL < 0.1ppm)	Superfund - CERCLA

APPENDIX D

MODELLING ASSUMPTIONS FOR GENERATION OF SOIL CONTOUR MAPPING USING SURFER

Appendix D: Modelling Assumptions for Generation of Soil Contour Mapping Using SURFER

Concentration contour maps were produced using the graphics program **SURFER version 4.0**. The contour maps were only produced where there was a difference between the minimum and maximum values of greater than twice the minimum value (Jones, R. 1994. personal communication). These maps are only statistical approximations of the spatial distribution of the different contaminants. The maps are only to be used to provide information on the approximate areas and/or patterns of contamination. They cannot be used to determine the actual concentration of a contaminant at a location where samples were not taken.

The contours produced by the program are significantly affected by the spatial distribution of the sampling sites, the accuracy of the positional information for the sampling sites, and the program options used to generate the contours. As the contours are affected by the spatial distribution of the sampling sites, the accuracy of the contours falls off at the edges of the map and in large areas where there are no sampling sites. Contours near the edge of the map should be interpreted with caution. Large areas without sampling sites have been blocked out so no contours were drawn through them.

The program options used in generating the contours remained constant for each map. The options used for the maps in this report are given below.

Grid Interpolation Method:	Kreiging
Search Method:	Normal
Search Radius:	Full Width of Map
No. of Nearest Points:	10
Grid Size:	26 x 20
Contour (in line) Smoothing:	Yes
Tension Factor:	2

The contour interval, minimum contour, and maximum contour for each map are given at the bottom of each map. The units of measurement for the contours are the same as in the tables. The location of the sampling sites are indicated by an asterisk and the approximate locations of major features such as roads are shown.

APPENDIX E

MERCURY CONCENTRATION IN SURFACE SOIL COLLECTED IN THE VICINITY OF ICI, CORNWALL - 1976, 1978, 1985, AND 1991

ppendix E: Mercury Concentration (µg/g, dry weight)* in Surface Soil (0-5 cm) Collected in the Vicinity of ICI, Cornwall - 1976, 1978, 1985 and 1991

STATION NUMBER	DISTANCE (M) AND DIRECTION FROM HG CELL ROOMS	1976 AUG.	1978 AUG.	1985 AUG.	1991 MAY	1991 AUG.
1	360 N	2.10	2.20	2.60	1.07	1.55
2	360 NNE	2.10	4.30	3.30	1.70	1.85
4	240 E	2.60	4.20	3.70	1.75	1.95
6	100 ENE	0.93	1.30	0.43	0.84	0.81
8	560 E	0.26	0.27	0.48	0.39	0.38
9	600 ENE	0.56	0.60	0.97	0.36	0.27
10	750 NNE	0.28	0.31	0.51	0.29	0.27
11	1120 NE	0.27	0.30	0.26	0.39	0.43
12	680 NW	0.05	0.02	0.06	0.06	0.04
14	240 SW	0.40	0.61	0.79	0.60	0.83
16	760 ESE	0.14	0.20	0.34	0.18	0.17
19	760 SW	NS	0.33	1.40	0.23	0.23
21	720 SSE	0.19	0.28	0.46	0.48	0.67
22	1700 E	NS	0.14	0.26	0.18	0.15
23	760 N	0.07	0.14	0.12	0.07	0.06
25	1400 NNE	0.23	0.15	0.32	0.14	0.20
27	840 E	0.48	0.23	0.36	0.22	0.22
32	1880 ENE	0.15	0.13	0.20	0.60	0.57
33	700 W	0.08	1.75	0.15	0.11	0.08
35	1740 WSW	0.08	0.06	0.09	0.11	0.08
36	2300 NW	NS	0.14	0.12	0.07	0.06
37	3500 NE	NS	0.08	0.09	0.07	0.05
Mean soil Hg concentration		0.59	0.81	0.77	0.45	0.50
Mean concentration in rural control soil samples				0.07	0.05	0.04
% of stations exceeding ULN**		28	32	32	27	32

Average of triplicate sample results in 1976 and 1978 and the average of duplicate sample results in 1985 and 1991.

Phytotoxicology section "Upper Limits of Normal" mercury concentration in urban soil (0-5 cm depth) is 0.5 µg/g (see appendix). Data exceeding the ULN are under lined.

; No sample.

urce: Dixon and Emerson, 1994

APPENDIX F

HUMAN SOIL INGESTION RATES

Appendix F: Human Soil Ingestion Rates

I. Overview of Soil Ingestion Rates

Faced with relatively little research evidence, it has been traditional to assume or crudely estimate a value for the amount of soil/dust ingested by young children. Typical average ingestion rates of this type range from 50 to 250 mg/day for children 2-3 years old (Hawley, 1985). A number of documents have used an average estimate of 100 mg of dirt ingested daily by children to represent a probable value for a "typical" child. Children suffering from pica may ingest more than 1 g/day (NAS, 1980).

Biokinetic modelling studies of lead intakes have suggested that 50 to 60 mg/day for ingestion of soil and dust is a more accurate estimate (Huffnage, 1988).

Recent progress in examining this question has involved the utilization of epidemiological mass balance studies in young children (Clausing *et al.*, 1987, Binder *et al.*, 1986; Calabrese *et al.*, 1989, 1990; Davis *et al.*, 1990; van Wijnen *et al.*, 1990. The most comprehensive of these are the Calabrese *et al.*, 1989 and Davis *et al.*, 1990 studies. These studies essentially measure the fecal excretion of relatively non-absorbed tracer elements found in soil (including aluminum, silicon, zirconium and titanium). These measurements are then utilized in mass-balance equation to derive an estimate of soil intake. Various tracers yield a range of estimates both within and between studies.

The principle finding of the 1989 Calabrese *et al.* study were that the median soil intake value based on all eight tracers ranged form 9 to 96 mg/day, with seven of the eight tracers having median values less than 50 mg/day. The three most reliable tracers revealed a range of median values of from 9 to 40 mg/day of soil ingested. The Davis *et al.* work indicated median daily estimates of 25.3 mg/day based on aluminum, 59.4 mg/day based on silicon and 81.3 mg/day based on titanium. Mean values were higher at 38.9, 82.4 and 245.5 mg/day for Al, Si, and Ti respectively.

Estimates based on titanium are typically highly variable and may be as much as an order of magnitude larger than those with other tracers. Calabrese has concluded that Ti estimated values in studies which lack food ingestion data may significantly overestimate soil ingestion (4-6X for Calabrese data if mass-balance approach not used). The authors conclude that the discrepancy is largely based on high levels of Ti in food and that the three most reliable tracers are Al, Si, and Y based on validation in adult volunteers. It has been suggested that the titanium-based estimates be treated as outlying and given less weight (U.S. EPA, 1989).

From this information a value of 80 mg/day soil ingested for children is selected for the exposure estimates for this pathway. There is no empirical data regarding soil ingestion rates for adults. A value of 20 mg/day is used, as adults will not have the same degree of hand-mouth activity. These values are in keeping with the recommended Canadian reference values for dirt, dust and soil intake (HWC, 1988).

II. 1992 Technical Memorandum - Analysis of Soil ingestion rates in Children

February 5, 1992.

MEMORANDUM

To: L. Hofmann
Chair,
CCME Subcommittee on Environmental Criteria for Abandoned Orphan Sites

From: S. Fleming
Senior Regulatory Toxicologist
Risk Assessment Unit
Hazardous Contaminants Coordination Branch

Subject: Evaluation of Soil Ingestion Rates for Children

Thank you for your invitation to review the draft soil ingestion values as suggested in the updated version of the Reference Values for Canadians document. My views have been formed through having examined and discussed this issue and the relevant scientific literature several times in the past few years. This has been primarily through my work on development of Ontario site-specific risk assessments of soil exposures on publicly accessible properties, the exposure modelling analysis for the development of multimedia guidelines for lead and as an invited reviewer of the EPA exposure methodologies for lead. The following is not a detailed evaluation of the literature but rather an attempt to bring some important scientific issues to your attention. It goes beyond review of the specific HWC documentation.

Of the areas of uncertainty in estimating soil-based risk (i.e concentration in soil vs. concentration as ingested, soil ingestion and bioavailability), soil ingestion rate probably has the least associated degree of uncertainty and it therefore is reasonable and useful, as Health and Welfare is undertaking, to attempt to fix a common value (or range) to assist in guideline development purposes. However, unlike drinking water consumption, for which we have remarkably good data, the database for this parameter is very limited and therefore considerable uncertainty remains from a scientific perspective. I therefore believe it is important for your Committee's specific purposes of setting soil guidelines to examine this question in more detail outside of the Reference Value document, which deals with soil ingestion amongst a host of other reference values. The interpretation of these soil studies is a rather complex matter, and given the important guidance being developed by your group, requires a more robust discussion.

In considering the quantitative information on which to base a crude estimate of soil exposure it is important to consider that very few studies (as outlined in the HWC draft)

have been conducted to shed light on this question. It is my view that either individually or collectively, there is insufficient study to arrive at a soil ingestion rate for children with confidence. In other words, these studies are helpful, but more study is required and we are still left with making an assumption. Therefore, what value should be chosen and what is the best information on which to base this choice?

The explanation provided for the choice of 50 mg/day is based upon the pooling of average results of selected studies with the Davis *et al.* (1990) study being omitted. The difficulty with this presentation is that it does not present a critical assessment, quantitatively or quantitatively of the available studies.

There are several problems with this:

- all of the studies suffer from some critical methodological limitations, which could be argued to invalidate the conclusions. Take for instance the Binder *et al.* (1986) study. No intake measures are made of tracer contribution from food and other nonsoil soils, and fecal sample loss was not quantified. Again, in the Clausing *et al.* (1987) and van Wijnen *et al.*, (1990) studies, no food was collected from non-institutionalized subjects. These studies are therefore unable to utilize a true mass-balance methodology. Further, the van Wijnen study did not assure total daily fecal collections. The arbitrary assumptions regarding fecal weight can greatly bias estimates of soil ingestion values. The use of the least tracer method (LTM) by van Wijnen *et al.* (1990) is likely to underestimate the rate of soil ingestion. There are many other examples. Therefore, the exclusion of the Davis *et al.*, (1990), which can be argued in several ways to be a superior study with less significant methodological limitations, is not warranted.

- I would conclude that of the studies available, the Calabrese *et al.* (1989) and Davis *et al.* (1990) provide the best available data in that they utilize soil tracer methodology incorporating a mass-balance approach. This is generally seen as the method from which to make reliable estimates of soil ingestion. It should be pointed out that while food collection is an absolutely essential component of this type of study, it is not sufficient to eliminate food as a potentially large contributor to variability in soil ingestion. A further difficulty has been recently pointed out by Dr. Calabrese, that the soil ingestion values from both studies may be far below the level of detection for the tracers. This would seriously question the usefulness of even these estimates.

- The questions of which tracers are reliable is crucial. This can be assessed either biologically or statistically. Each tracer gives a different mean, median and range of soil ingestion rates (see Table). Ideally a tracer will be poorly absorbed, have little potential for inhalation exposure and be present in indigestible items in only trace amounts. Some have argued that Al and Si are preferred, while others suggest Zr and Y may be better. This indicates the need to explicitly describe the results and variation with each tracer.

- if one looks at specific tracers like Al and Si, Davis *et al.* (1990) report means of 38.9 (Al) and 82.4 (Si) mg/day. Calabrese *et al.* (1989) report mean values for combined soil and dust ingestion of 154 (Al) and 483 (Si) mg/day. Mean values for soil alone were 153 (Al) and 154 (Si) mg/day. The HWC document seems to "average" the range of the median values (soil alone) for reliable tracers (Al, Si, Y). This raises the question of the use of (average) versus median estimates. Some have argued that the mean is more appropriate in developing protective measures for populations.

Having outlined the above considerations, I am concerned that the proposed default assumption for soil ingestion being suggested may be too low and thereby tend to underestimate soil-based risks. Barring technical/economic considerations, if applied directly this may lead to development of insufficiently stringent guidelines. Remembering that the current database is weak, we are still left with assuming a value (or range) on this parameter. We should also remember that many children (non-pica) consume much more soil/dust than a "typical" child. I have previously utilized values of 80 or 100 mg/day in my exposure analyses. This value is at worst slightly conservative for a typical child and may greatly underestimate the exposure for higher risk children. I would feel most uncomfortable in applying a value which is roughly 50% less than this.

Your Committee may also wish to consider the question of the use of the "average" receptor in the case of children. For example, with lead, our analysis has suggested that upwards of 50% of typical daily intake for children may be soil/dust based, thereby making this a critical pathway. For carcinogenic organics, the childhood period (0.5-4 years) represents only a fraction of the lifetime exposure, however for substances like lead, this period of life is critical and shapes our regulatory efforts. There may therefore be some argument for applying the upper 95% confidence interval of soil ingestion rates to account for all children. Calabrese has reported a range of median values of 0 to 142 mg/day covered by the 95 % CI for the five tracers with smallest relative variance.

In summary, I would recommend that values that contain some measure of conservatism continue to be applied for soil ingestion rates for children (for example, 80-100 mg/day). As well, in my view, some analysis (science and policy) of the upper 95% CI for exposure is warranted. Lastly, as I have previously suggested to you, I would recommend that, if feasible, a mass-balance study of soil ingestion in a group of Canadian children be sponsored. This would provide additional information specifically for Canadian children and would be a valuable contribution to the small number of international studies in this area. This may allow a reassessment of this information at a later date.

I hope this provides you with the type of comment which you and your Committee were seeking on this question. I trust you will ensure that Dr. Sitwell and others are provided these comments directly. I would be most happy to discuss any of these matters with you or your committee members.

Daily Intake of Soil and Dust Estimated from Elemental Abundances

Study	Lead Intake (mg/day)			
	Element	Median	Mean	Maximum
Davis *et al.* (1991)	A1	25	39	904
	Si	59	82	535
	Ti	81	246	6182
Calabrese *et al.* (1989)	A1	30	154	4929
	T1	30	170	3597
	Y	11	65	5269
	Zr	11	23	838
Binder *et al.* (1986)	A1	121	181	1324
	Si	136	184	799
	Ti	618	1834	17076
Clausing *et al.* (1987)	A1	92	232	979
	Ti	269	1431	11620

Soil and Dust Intake Depends on the Environment

Sampling Group	Age	Geometric mean intake (A1,Si,Ti)
Day care centre (good weather)	1-2	33 - 88
Day care centre (rainy weather)		0 - 19
Campgrounds		150 - 200
Day care centre (good weather)	3+	12 - 62
Day care centre (rainy weather)		0 - 29
Campgrounds		31 - 81

APPENDIX G

SPORT FISH CONTAMINANT MONITORING PROGRAM AND DERIVATION OF FISH CONSUMPTION ESTIMATE FOR ANGLERS

Appendix G: Sport Fish Contaminant Monitoring Program and Derivation of Fish Consumption Estimate for Anglers

The Ontario Government monitors contaminant levels in sport fish through the Sport Fish Contaminant Monitoring Program. These monitoring efforts are the basis of consumption advice incorporated into the annual "Guide to Eating Ontario Sport Fish". In 1992, a questionnaire was included in the Guide to get a sense of the effectiveness of the consumption advice, fishing frequency, the most fished locations and the fish consumption patterns. For the purposes of the exposure analysis, the fish consumption figure of 22.5 g/day derived from this questionnaire is used to estimate the exposure of anglers as this is the most recent data collected as well as being relevant to the region in which Cornwall is part.

Of the 494 replies received, 98% of the respondents resided in Southern Ontario and 24.4% fished more than once per week and only 4.6% fished in the St. Lawrence River. Walleye was the most frequently caught and consumed sport fish (>66%). Over 25% of the respondents indicated that they caught and consumed fish approximately once per month with greater than 60% consuming fish at least once per month.

Calculations used for Sport Fish Consumption Figures

The following methods were used to calculate the sport fish consumption figures used in this report. As well, these methods were applied to determine the commercial fish consumption figures.

a) **Mean Meal Size**

The mean meal size was calculated from responses to question 11 in the questionnaire, which asked "How much fish caught by angling from Ontario waters do you eat at a single meal?" The calculations were done initially in ounces, and for the "less than 60 g (2 oz)" response, a value of one ounce was assumed. As well, for the "more than 450 g (1 lb)" response, 20 ounces was assumed. The number of responses for each size category was determined and the results were totalled. The number of respondents who did not consume any sport fish were deleted from the calculations. The total was divided by the number of fish-consuming respondents to derive an average meal size of 9.7 ounces or 276 grams.

b) **Number of Sport Fish Meals Consumed/Year**

The number of sport fish meals consumed/year was determined from the responses to question 10, which asked "How often did you eat these fish in 1991?" All the responses were converted to a number of days out of the year (e.g., "daily" = 365, once/week = 52, etc.) For the "greater than once/week" response, three times/week (156) was assumed, and numbers were used, as given, in the "on

vacation only", "__ times" and "other" categories. The number of responses for each category, multiplied by the equivalent number of days, was totalled and divided by the number of respondents who consumed sport fish. This gave an average figure of 29.8 or approximately 30 meals/consumer/year.

c) **Daily Sport Fish Consumption Figure**

This figure is derived from the following calculations:

number of meals/consumer/year (29.8) X mean meal size (9.72 oz) = number of ounces consumed/year (289.7)

This result was converted to grams/year (multiplying by the conversion factor of 28.35), and was divided by 365 days to give a daily sport fish consumption figure of 22.5 g.

APPENDIX H

DRINKING WATER SURVEILLANCE PROGRAM RESULTS FOR MERCURY FROM 1990 TO 1993

Appendix H: Drinking Water Surveillance Program results for mercury from 1990 to 1993 (µg/L)

YEAR	MONTH	RAW		TREATED	
CHARLOTTENBURG WATER TREATMENT PLANT					
1991	May	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	July	0.02	<W	0.02	<W
	August	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	October	0.02	<W	0.02	<W
	November	0.02	<W	0.02	<W
1992	January	0.02	<W	0.02	<W
	February	0.02	<W	0.02	<W
	March	0.02	<W	0.02	<W
	April	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	August	0.02	<W	0.02	<W
	October	0.02	<W	0.02	<W
	December	0.02	<W	0.02	<W
1993	February	0.18		0.02	<W
	April	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	August	0.02	<W	0.02	<W
	October	0.02	<W	0.02	<W
	December	0.02	<W	0.02	<W
CORNWALL WATER TREATMENT PLANT					
1990	May	0.02	<W	0.02	<W
	July	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	November	0.02	<W	0.02	<W
1991	January	0.02	<W	0.02	<W
	March	0.02	<W	0.02	<W
	May	0.02	<W	0.02	<W
	July	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	November	0.02	<W	0.02	<W
1992	March	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	December	0.02	<W	0.02	<W
1993	May	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W

YEAR	MONTH	RAW		TREATED	
PRESCOTT WATER TREATMENT PLANT					
1990	May	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	July	0	ISS	0.02	<W
	August	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	October	0.02	<W	0.02	<W
	November	0.02	<W	0.02	<W
	December	0.02	<W	0.02	<W
1991	January	0.02	<W	0.02	<W
	February	0.02	<W	0.02	<W
	March	0.02	<W	0.02	<W
	April	0.02	<W	0.02	<W
1991	May	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	July	0.02	<W	0.02	<W
	August	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	October	0.02	<W	0.02	<W
	November	0.02	<W	0.02	<W
1992	March	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	December	0.02	<W	0.02	<W
1993	March	0.02	<W	0.02	<W
	June	0.02	<W	0.02	<W
	September	0.02	<W	0.02	<W
	December	0.02	<W	0.02	<W

<W = less than the detection limit
ISS = Sample not preserved properly

APPENDIX I
DERMAL ABSORPTION OF INORGANIC IONS

Appendix I: Dermal Absorption of Inorganic Ions

Human skin is made up of three layers: the stratum corneum (10 μ), viable epidermis (100 μ) and the papillary layer of the dermis (100-200 μ). The permeability of the capillary walls to other than macromolecules is sufficiently great that diffusing molecules are readily absorbed into the capillary circulation. Hair follicles and sweat glands pierce this layer. However, their contribution to absorption of molecules and ions is probably negligible, except in the initial phase of absorption.

The stratum corneum is the main barrier to diffusion across the skin. This has been demonstrated experimentally. It is formed from dried, compact keratin-containing cells that are converted from aqueous epidermal cells. It is always partially hydrated and varies in thickness on the body.

The skin barrier can be regarded as a composite membrane, pierced over a small portion of its area by shunts of different but lower diffusivities. For a simple membrane, the steady-state flux of solute, J_s is given by:

$$J_s = K_m D \triangle C_s / \delta$$

where K_m is the solvent-membrane distribution coefficient, $\triangle C_s$ is the concentration difference of solute across the membrane, D is the membrane diffusion coefficient for the solute and δ is the membrane thickness. The permeability coefficient, k_p, is defined as $K_m D/\delta$.

The permeability constant for water at 25°C is approximately 0.5×10^{-3} cm/hr, corresponding to a flux of 0.2 mg/cm^2/hr. The flux is approximately the same whether liquid water or saturated water vapour is applied in vitro.

Electrolytes in aqueous solution do not penetrate the skin as readily as water because:

- the stable hydration sphere around an ion makes it a much larger diffusing unit than a water molecule;
- the charge on the ion will interact with co-ions, counter-ions and fixed charges in the tissue.

Therefore, it is likely that, in the absence of a potential gradient, most ions will penetrate much more slowly than water, and no ion will penetrate faster. Measured permeability constants (k_p) are approximately same for many ions and are of the order of 10^{-6} cm/hr. Shunt diffusion through appendages may play a significant role.

APPENDIX J

MODELLING POTENTIAL MERCURY EMISSIONS FORM CONTAMINATED SOILS

MEMORANDUM February 28, 1995

TO: Scott Fleming
Sr. Regulatory Toxicologist
Standards Development Branch

FROM: Rob Bloxam
Program Leader; Atmos. Modelling
Science & Technology Branch

SUBJECT: Modelling Possible Mercury Emissions From Contaminated Soils Near ICI (Cornwall) and Maximum Air Concentrations Due to Those Emissions

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To calculate the possible air concentrations due to re-emission of mercury from contaminated soils near the ICI plant in Cornwall, the emission rates must first be estimated. Since the mercury in soils is in a sulphite form with very low volatility it must first be converted back to elemental mercury by biological processes. This biological breakdown of the mercury sulphite likely depends on temperature and sunlight. The elemental mercury can then volatilize at a rate which also depends on temperature. Since we have no information on the rate constants for the biological degradation of the mercury sulphite, I have attempted to estimate the maximum likely emission rate using mercury concentrations measured over the site of the contaminated soils.

A TAGA survey of the ICI facility in Cornwall was conducted in June 1993. Their primary objective was to measure mercury concentrations downwind of the ICI plant but they also performed some upwind measurements at the beginning and end of each days measurements. The 'upwind' site usually used in this survey was very close to the ICI building and part of the observed concentrations could still be coming from direct emissions from the building during light wind conditions. In addition, the measured concentrations would include global background mercury concentrations which are about 1.5 ng/m^3. A description of this TAGA survey along with the measured concentrations is given in a Technical Memorandum from Andy Ng to Maris Lusis dated July 21, 1993. Half hour average mercury concentrations measured at the upwind site ranged from 1.9 to 9.3 ng/m^3 on June 16,17,23 and 24. Much higher concentrations in the hundreds of ng/m^3 were observed downwind of the plant.

The maximum likely mercury emission rates were derived by back calculating the emission required from the soils to account for the observed concentrations when the TAGA was upwind of the ICI plant. The attached Figure 1 shows measured soil concentrations around the plant. For modelling purposes the land area above about 0.5 (µg Hg/g soil) was used as the source region of mercury volatilizing from soils. This source region was subdivided into 25 area emission sources as shown on Figure 1. The emission rate was assumed to be uniform across this area. It was conservatively assumed that the measured mercury concentrations were due entirely to emissions from the contaminated soils. Observed meteorological conditions during the 8 half hour periods for which back calculations could be performed were obtained from the TAGA report and from surface weather maps for this area. The dispersion model used in the calculations was the MOEE CAP model (Appendix 8 of MOEE Clean

Air Program document, 1990).

The back calculations for 4 morning upwind measurements gave emission estimates ranging from 0.5 to 1.7 x 10^{-3} g/s while the 4 afternoon upwind measurements resulted in a range from 1.1 to 3 x 10^{-3} g/s. The somewhat higher emission estimates in the afternoon might in part be due to higher temperatures. The above emission rates can then be applied during the meteorological conditions giving the least dispersion and therefore the maximum concentrations for the residential community outlined on Figure 1. An emission rate of 3 x 10^{-3} g/s was used to estimate the maximum likely concentrations due to emissions of mercury from these contaminated soils.

Using the above emission rate, the model was run using the worst meteorological condition to estimate the maximum likely air concentrations over the residential community marked on Figure 1. The meteorological condition that would result in maximum concentrations is a very light wind speed, during stable conditions. The highest calculated concentrations using the conservative emission estimate and the worst meteorological conditions was about 400 ng/m^3. This could be a very conservative number since the worst case meteorological condition would only occur at night or during the winter when mercury emissions from soils could be minimal (i.e., the biological degradation of mercury sulphite depends on temperature and is thought to require sunlight).

A second calculation was performed to estimate the typical concentrations which could occur over the residential area due to mercury emissions from soils. The emission rate used in this case was the average of the eight half hour average values (i.e., 1.5 x 10^{-3} g/s). This could still be an overestimate of the emission rate since the observed concentrations were assumed to be entirely due to soil emissions when estimating the emission rates. For this calculation the model was run for 'typical' meteorological dispersion conditions using wind from all directions. The typical meteorological condition used was a wind speed of 4 m/s with neutral stability. The highest modelled concentrations for any given hour with this meteorological condition were about 20 ng/m^3. Averaging over all wind directions results in an impact of from 3 to 9 ng/m^3 at locations across the residential area examined.

If the emission rates described above were to continue at a constant rate throughout the year, the total mercury emissions and the time required to volatilize all of the mercury contamination can be estimated. A total of about 50 kg of mercury would be released in a year if an emission rate of 1.5 x 10^{-3} g/s were used. Since the total mass of mercury to a depth of 1 meter over the 1.5 km^2 contaminated area is about 4000 kg, it would take ≈80 years to volatilize the mercury to a depth of 1 meter.

Robert Bloxam

cc Dr. N. Reid

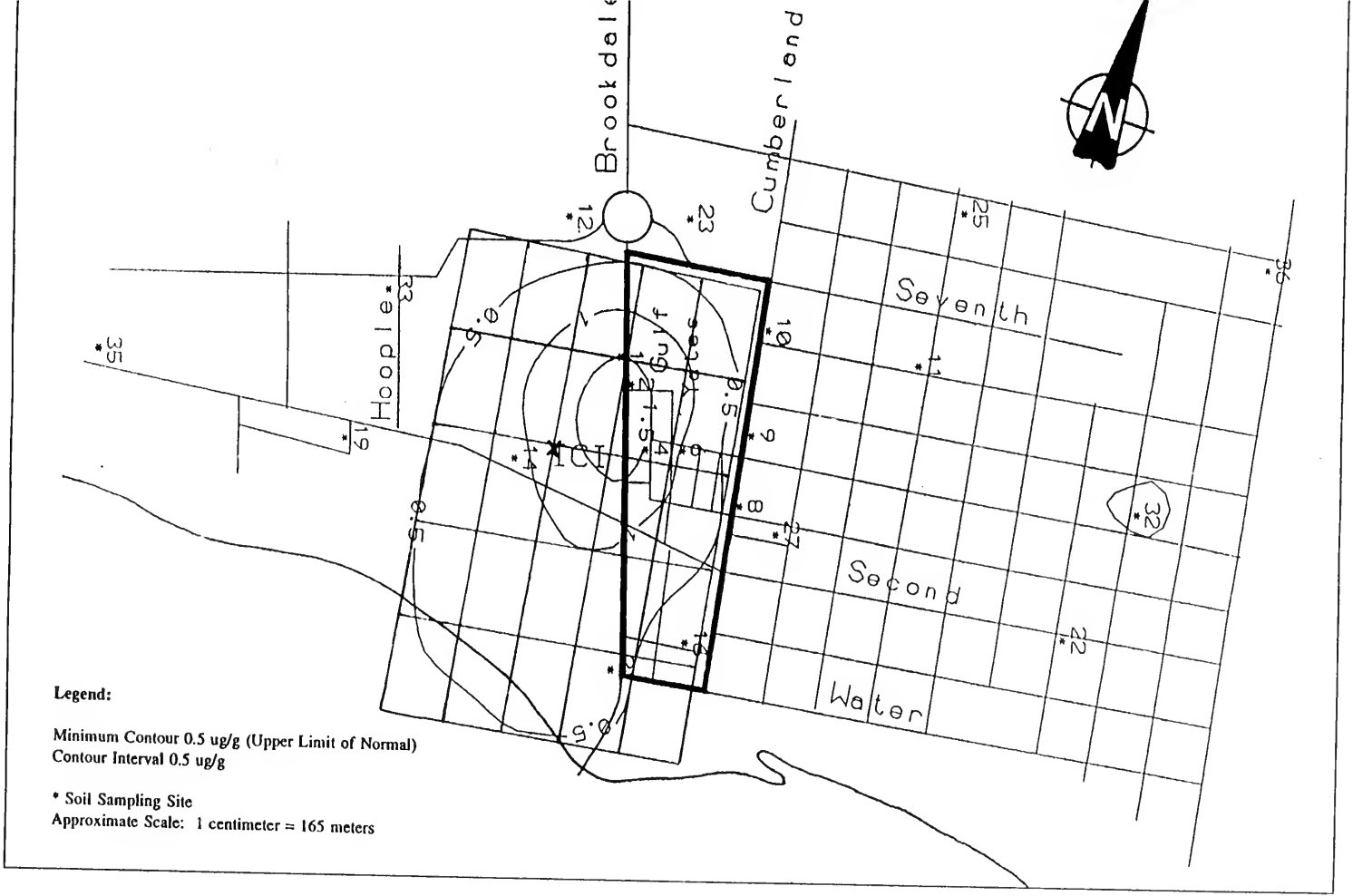

Figure 1: Mercury in surface soil: Cornwall - 1991 (Contours produced using SURFER)

APPENDIX K

MERCURY CONCENTRATION IN SOIL (TRIPLICATE SAMPLES) FROM CORNWALL, IN PPM DRY WEIGHT 1976-1978 AND RESPECTIVE MEANS - DEPTH PROFILES

Appendix K: Mercury Concentrations in Soil (Triplicate Samples) From Cornwall, in ppm dry weight 1976-1978 and Respective Means - Depth Profiles

STATION	1976		1977		1978		Mean	
	0-5 cm	5-10 cm	0-5 cm	5-10 cm	0-5 cm	5-10 cm	0-5 cm	5-10 cm
1	2.1	0.66	2.0	0.52	2.2	0.56	2.1	0.38
2	2.1	0.59	3.2	0.47	4.3	1.5	3.2	0.85
3	5.1	3.1	5.6	3.0	2.1	1.1	4.3	2.4
4	2.6	0.46	2.8	0.27	4.2	1.2	3.2	0.64
5	1.8	1.0	2.5	0.35	2.2	1.6	2.2	1.25
6	0.93	0.54	1.2	0.43	1.3	0.71	1.1	0.56
7	0.96	0.50	0.56	0.36	0.55	0.44	0.69	0.43
8	0.26	0.18	0.38	0.17	0.27	0.17	0.91	0.17
9	0.56	0.47	0.59	0.59	0.50	0.48	0.58	0.51
10	0.28	0.19	0.41	0.19	0.31	0.22	0.33	0.20
11	0.27	0.29	0.45	0.37	0.30	0.30	0.34	0.32
12	0.05	0.03	0.07	0.05	0.02	0.02	0.05	0.03
13	0.87	0.89	0.88	0.30	1.0	0.71	0.92	0.7
14	0.40	0.34	0.50	0.22	0.51	0.32	0.50	0.29
15	0.50	0.40	0.56	0.36	0.51	0.34	0.57	0.37
16	0.14	0.13	0.51	0.36	0.20	0.22	0.28	0.31
17	0.52	0.17	0.59	0.23	0.32	0.43	0.46	0.26
18	0.48	0.36	0.34	0.22	0.46	0.25	0.43	0.28
19					0.33	0.16	0.33	0.16
20	0.35	0.28	0.31	0.07	0.39	1.02	0.35	0.46
21	0.19	0.08	0.16	0.06	0.28	0.18	0.21	0.11
22					0.14	0.14	0.14	0.14
23	0.07	0.06	0.12	0.09	0.14	0.12	0.11	0.05
24	0.08	0.10	0.20	0.18	0.15	0.12	0.14	0.13
25	0.23	0.21	0.14	0.16	0.15	0.10	0.17	0.15
26	0.66	0.62	0.33	0.26	0.43	0.44	0.47	0.44
27	0.48	0.44	0.30	0.16	0.23	0.20	0.34	0.27
28	0.33	0.26	0.20	0.17	0.26	0.24	0.25	0.23

STATION	1976		1977		1978		Mean	
	0-5 cm	5-10 cm	0-5 cm	5-10 cm	0-5 cm	5-10 cm	0-5 cm	5-10 cm
29	0.04	0.03	0.05	0.06	0.06	0.06	0.05	0.04
30	0.04	0.04	0.05	0.03	0.06	0.06	0.05	0.04
31	0.23	0.21	0.25	0.16	0.23	0.20	0.24	0.19
32	0.15	0.13	0.22	0.19	0.13	0.14	0.17	0.20
33	0.08	0.06	0.36	0.68	1.75	1.02	0.22	0.58
34	0.07	0.05	0.05	0.02	0.07	0.05	0.06	0.04
35	0.08	0.05	0.04	0.04	0.06	0.05	0.06	0.03
36					0.14	0.08	0.14	0.08
37					0.08	0.06	0.08	0.06
38					0.12	0.08	0.12	0.08
39					0.19	0.21	0.19	0.21
40	0.04	0.04	0.03	0.04	0.05	0.06	0.03	0.06

Source: **Bob Emerson, personal communication**